MAGNESIUM SULPHATE ADDED AS AN ADJUVANT TO INTRATHECAL BUPIVACAINE IN PATIENT WITH MILD PREGNANCY INDUCED HYPERTENSION UNDERGOING CAESAREAN SECTION

Dissertation submitted
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CERTIFICATE

This is to certify that this dissertation titled **"MAGNESIUM SULPHATE ADDED AS AN ADJUVANT TO INTRATHECAL BUPIVACAINE IN PATIENT WITH MILD PREGNANCY INDUCED HYPERTENSION UNDERGOING CAESAREAN SECTION"** is a bonafide work done by Dr.K.Venkatesan under my supervision in the Department of Anesthesiology, **Government Kilpauk Medical College and Hospital** , Chennai during the academic period 2009-2012 and is being submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfillment of the University regulation for the award of Medicine (M.D Anesthesiology and critical care) his dissertation is a bonafide work.

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DECLARATION

I, **Dr.K.Venkatesan** solemnly declare that the dissertation, **"MAGNESIUM SULPHATE ADDED AS AN ADJUVANT TO INTRATHECAL BUPIVACAINE IN PATIENT WITH MILD PREGNANCY INDUCED HYPERTENSION UNDERGOING CAESAREAN SECTION"** is a bonafide work done by me in the Department of Anesthesiology and Critical care, Government Kilpauk Medical College, Chennai under the guidance of **Prof.Dr.P.S.Shanmugam, M.D.,D.A.,** Professor and HOD, Department of Anesthesiology, Government Kilpauk Medical College, Chennai.

Place: Chennai Signature

Date: **(Dr.K.VENKATESAN)**

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CONTENTS

ABSTRACT

Title: Magnesium sulphate added as an adjuvant to intrathecal Bupivacaine in patient with mild pregnancy induced hypertension undergoing caesarean section

Author: prof. P.S. Shanmugam M.D.D.A

Back ground: Adequate analgesia following caesarean section decreases Morbidity, Ambulation, Improve patient outcome and facilitate care of the newborn baby .Intrathecal Magnesium an NMDA antagonist has been shown to prolong analgesia without significant side effect in healthy parturients. we therefore studied the effect of adding intrathecal Magnesium sulphate to Bupivacaine , Fentanyl in patient with mild pregnancy induced hypertension undergoing caesarean section.

Materials and Methods: After obtaining ethical committee approval from Govt. Kilpauk medical college ,60 patients with ASA I &II between the age group of 18 -35 undergoing elective caesarean section under spinal anesthesia were randomly divided in to three groups.

Group C –Control group, (N=20) patients 0.5% 2cc (10mg) Bupivacaine +0.6cc normal saline

Group F-Fentanyl group, (N=20) patients 0.5% 2cc (10mg) Bupivacaine+0.5cc (25micgm) Fentanyl+0.1cc normal saline

Group M -Magnesium sulphate group, (N=20) patients 0.5% 2cc (10mg) Bupivacaine+0.5cc (25micgm) Fentanyl+0.1cc 50% (50mg) magnesium sulphate.

Onset , Duration and recovery of sensory and motor block duration of spinal anesthesia, APGAR score and post operative analgesia duration were studied.

Results: Onset of sensory and motor blockade was delayed in the magnesium sulphate group duration of spinal anesthesia and motor block duration is prolonged in Magnesium sulphate group.(189.40 minutes) post operative analgesia was significantly prolonged in the Magnesium sulphate group when compared to control group(403.65vs 222.45minutes) hemodynamic parameter at 1,5,10,15,20,30 min were evaluated.

Conclusion: There is delay in onset of sensory and motor blockade with use of Magnesium sulphate. However there is prolonged motor blockade and duration

of analgesia overlaps well in to the post operative period .This is beneficial for the patient for post operative analgesia. APGAR Score not affected between the groups.

Key words: Fentanyl, Magnesium sulphate, Bupivacaine, Pregnancy induced hypertension

INTRODUCTION

Spinal anesthesia was first performed by **August Bier** on 16th August 1898 when he injected 3ml of 0.5% cocaine intrathecally. Spinal Anesthesia is simple, easy to perform and has got a definite endpoint. It requires a small dose of local anaesthetic drugs produces profound sensory and motor blockade. Ever since the introduction of local anaesthetic drugs diverse classes of drugs such as epinephrine, Opioids, Clonidine, Neostigmine, Ketamine and Benzodiazepines have been added as adjuvants to local anaesthetics in an attempt to prolong analgesia and reduce the incidence of side effects.

Magnesium has been called “Nature’s physiological calcium channel Blocker”.Parenteral magnesium has been used for many years on an empirical basis for intraoperative and postoperative analgesia. Although systemic magnesium decreases postoperative Opioid requirements, its intrathecal use has not been evaluated clinically. However, it has been safely used in humans and its safety profile has been documented in experimental studies.

In 1906, Haubold and Meltzer showed that intrathecal administration of Magnesium Sulphate produces spinal anesthesia that includes profound motor and sensory blockade without any permanent untoward effects.

In this prospective randomized double blind controlled study, we evaluated the effect of adding intrathecal Magnesium Sulphate to Bupivacaine and Fentanyl in patients undergoing elective Caesarean section.

AIM OF THE STUDY

To study and compare the effect of added Fentanyl 25 (micro gm) & $MgSO_4$ 0.1cc 50% (50mg) to 0.5% 2cc(10mg)Bupivacaine, in patients with mild gestational hypertension(PIH) undergoing elective Caesarean section under spinal anesthesia.

To evaluate

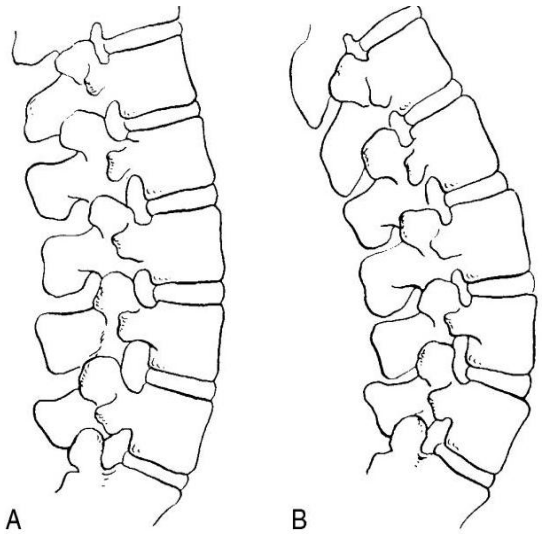
- Onset time of Sensory block
- Onset time of Motor block
- Upper level of analgesia
- Duration of postoperative analgesia
- Hemodynamic changes

APPLIED ANATOMY OF PREGNANCY

VERTEBRAL ANATOMY:

In women of child bearing age, the spinal cord terminates as conus medullaris at the level of the lower border of the first lumbar vertebral body. The conus medullaris is attached to the coccyx by means of neuro-fibrous band called the filum terminale, which is surrounded by the nerves of the lower lumbar and sacral roots known as cauda equina.

The subarachnoid space located between the pia mater and arachnoid mater, contains (1) Cerebrospinal fluid (CSF) (2) spinal nerves (3) Trabecular network between the two membranes (4) Blood vessel that supply the spinal cord and (5) The lateral extension of pia mater –the denticulate ligament.



Effect of pregnancy on lumbar spine: Fig A, Non pregnant. B, pregnant there is a marked increase in lumbar lordosis and a narrowing of the interspinous space during pregnancy.

The normal anatomic changes of pregnancy affect the use of neuraxial technique. Uterine enlargement and venacaval compression result in engorgement of epidural veins .The enlarged epidural veins also may displace cerebrospinal fluid(CSF) from the thoracolumbar region of the subarachnoid as does the greater intra abdominal pressure of pregnancy. This displacement of CSF and lower specific gravity of CSF, partly explains lower dose required for spinal anesthesia in pregnant patients.

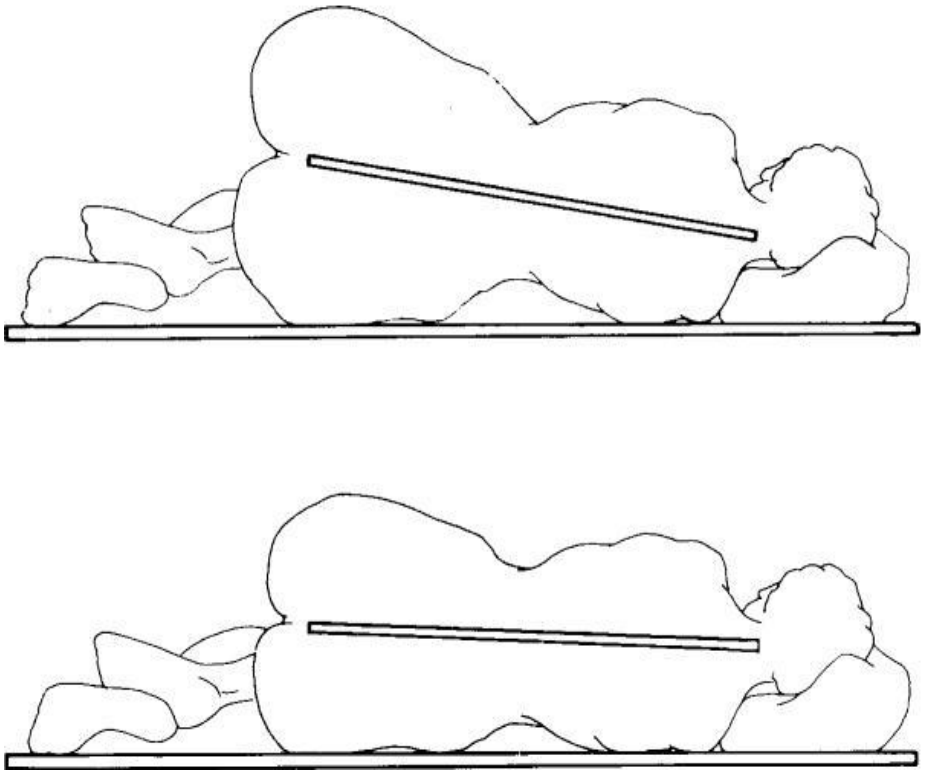
The hormonal changes of pregnancy affect the perivertebral ligamentous structures , including ligamentum flavum. The ligamentum flavum feels less dense and softer in pregnant women than in non pregnant.

Achieving flexion of the lumbar spine is difficult for pregnant women. Progressive accentuation of lumbar lordosis alter the relationship of surface anatomy to the vertebral coloum. The changes that may occur in pregnancy are

1. A pregnant women pelvis rotates on the long axis of the spinal column, thus the line joining the iliac crest assume a more cephalad relationship to the vertebral column.

2. There is less space between adjacent lumbar spinous processes during pregnancy. It may be more difficult to use the midline approach to identify the epidural or subarachnoid space in pregnant women.

3. MRI imaging has shown that the apex of the lumbar lordosis is shifted caudal during pregnancy, and the typical thoracic kyphosis in women is reduced during pregnancy. These changes may influence the spread of intrathecal anesthetic solution in supine patient



Pelvic widening and resultant head-down tilt in the lateral position during pregnancy

Anatomical changes in respiratory system:

The thoracic cage increases in circumference by 5 to 7 cm during pregnancy because of increases in both the anteroposterior and transverse diameters. Flaring of the ribs, which begins at the end of the first trimester, results in an increase in the sub costal angle from 68.5 to 103.5 degrees at term. The vertical measurement of the chest decreases by as much as 4 cm, which results from the elevated position of the diaphragm.

Capillary engorgement of the nasal and oropharyngeal mucosae and larynx begins early in the first trimester and increases progressively throughout pregnancy. Nasal breathing commonly becomes difficult, and epistaxis may occur because of nasal mucosal engorgement.

Airway conductance increases, indicating a dilation of the larger airways below the larynx. Factors contributing to airway dilation include the direct effects of progesterone, cortisone, and relaxin.

Anatomical changes in Gastrointestinal system:

The stomach is displaced upward toward the left side of the diaphragm during pregnancy, and its axis is rotated approximately 45 degrees to the right from its normal vertical position. The altered position of the stomach displaces the intra abdominal segment of the esophagus into the thorax . This causes a reduction in tone of the lower esophageal high-pressure zone (LEHPZ), which

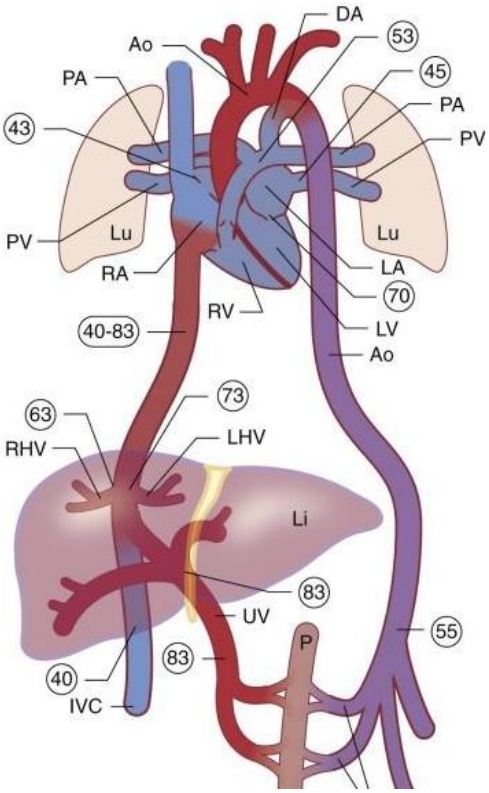
normally prevents the reflux of gastric contents. This displacement of the esophagus also prevents the rise in lower esophageal tone that normally accompanies an increase in intragastric pressure (IGP). Progestins also may contribute to a relaxation of the LEHPZ. IGP is elevated during the last trimester in all pregnant women. These anatomical changes predispose to increased risk of aspiration.

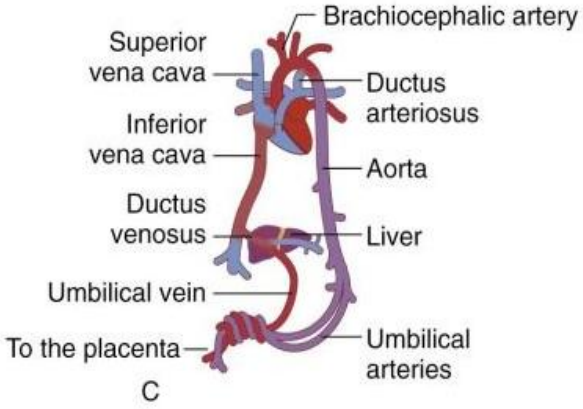
Feto-placental unit:

The placenta is composed of both maternal and fetal tissues that consist of a basal and a chorionic plate. It is a semi permeable membrane that provides an interface for the maternal and fetal circulation. The intervillous space separates the plates and is subdivided by decidual tissue. Chorionic villi and spiral arteries protrude into this intervillous space. Maternal blood flows into the intervillous space from the spiral artery while placental transfer from the mother to the fetus occurs. Approximately 80% of the uterine blood flow passes through the intervillous space.

Oxygenated blood leaves the placenta through fetal umbilical vein, enters the liver where flow divides between portal sinus and ductus venosus then empties into IVC. Inside the fetal heart, blood enters the right atrium through foramen ovale, where most of the blood is directed into left atrium and left ventricle, and then enters aorta. Blood is then sent to the brain and myocardium. Deoxygenated blood returning from lower extremities and SVC

is preferentially directed into right ventricle and pulmonary trunk, majority of blood passes through ductus arteriosus into descending aorta. Blood returns to the placenta through umbilical arteries for gas and nutrient exchange. Fetal blood flow is approximately 75 ml /kg/min, a rate far less than maternal flow.





APPLIED PHYSIOLOGY

PHYSIOLOGIC CHANGES OF PREGNANCY:

Maternal physiologic changes in pregnancy occur as a result of hormonal alterations, mechanical effects of the gravid uterus, increased metabolic and oxygen requirements, metabolic demands of the fetoplacental unit, and hemodynamic alterations associated with the placental circulation.

Cardiovascular system:

Cardiac output increases during pregnancy to meet the physiological demand. Increase in cardiac output is due to an increase in both stroke volume and heart rate, the more important factor is stroke volume, which increases by 20% to 50% at term.

Diastolic blood pressure falls to a greater degree than does systolic blood pressure. The decreased diastolic blood pressure is consistent with changes in systemic vascular resistance, which falls during early gestation, reaches its nadir (i.e., a 35% decline) at 20 weeks gestation, and rises during late gestation. However, systemic vascular resistance remains approximately 20% below the nonpregnant level at term. The decreased systemic vascular resistance results from the development of a low resistance vascular bed (i.e., the intervillous space) as well as vasodilation caused by prostacyclin, estrogens, and progesterone. Compression of the aorta by the gravid uterus may partly account for the greater systemic vascular resistance during the third trimester.

Central haemodynamics :

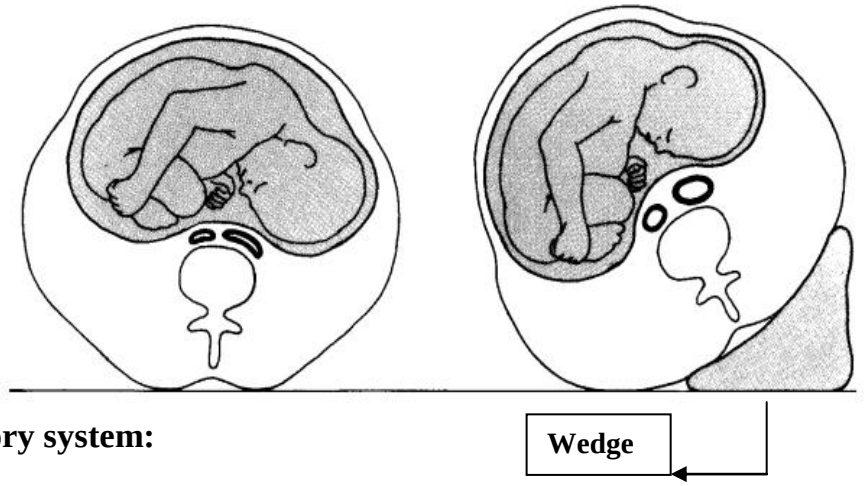
Parameter	Change
Cardiac output	+50%
Stroke volume	+20-30%
Heart rate	+20-50%
Left ventricular end-diastolic volume	Increased
Left ventricular end-systolic volume	No change
Ejection fraction	Increased
Left ventricular stroke-work index	No change
Pulmonary capillary wedge pressure	No change
Pulmonary artery diastolic pressure	No change
Central venous pressure	No change
Systemic vascular resistance	-20%
Blood Pressure	± 15%

Roll over test

Comparison of the blood pressure of a pregnant women lying on the backside, and excessive increase when she rolls to the supine position indicates increased risk of preeclampsia.

Aortocaval compression:

In supine position , gravid uterus compress the aorta and inferior vena cava leading to decreased venous return. Venous return occurs primarily by diversion of blood through the intraosseous vertebral veins, paravertebral veins, and epidural venous plexus. This drop in venous return for which the cardiovascular system cannot compensate could result in **supine hypotensive syndrome** .



Respiratory system:

Oxygen consumption increases by 30% to 40% during pregnancy. The progressive increase in oxygen consumption is caused primarily by the metabolic needs of the fetus, uterus, and placenta and secondarily by increased cardiac and respiratory work. Carbon dioxide production shows changes similar to those of oxygen consumption.

Respiratory mechanics:

Parameter	Change
Lung volumes	
Inspiratory reserve volume	+5%
Tidal volume	+45%
Expiratory reserve volume	-25%
Residual volume	-15%
Lung capacities	
Inspiratory capacity	+15%
Functional residual capacity	-20%
Vital capacity	No change
Total lung capacity	-5%
Dead space	+45%
Respiratory rate	No change
Ventilation	
Minute ventilation	+45%
Alveolar ventilation	+45%

Hematology:

Maternal blood volume begins to increase early in pregnancy as a result of changes in osmoregulation and the renin-angiotensin system, causing sodium retention and increasing total body water to 8.5 L. By term, blood volume increases by up to 45% whereas red cell volume increases by only

30%. This differential increase leads to the “physiologic anemia” of pregnancy with an average hemoglobin and hematocrit of 11.6 g/dL and 35.5%, respectively. However, oxygen transport is not impaired by this relative anemia because the mother's body compensates for it by increased cardiac output, increased Pa_{O_2}, and a rightward shift in the oxyhemoglobin dissociation curve. Hematologic parameters:

Parameter	Change or actual measurement
Blood volume	+45%
Plasma volume	+55%
Red blood cell volume	+30%
Hemoglobin	11.6 g/dL
Hematocrit	35.5%

Coagulation changes:

A state of hypercoagulability exists in pregnancy, with increased levels of most coagulation factors. Pregnancy is associated with enhanced platelet turnover, clotting, and fibrinolysis. Thus pregnancy represents a state of accelerated but compensated intravascular coagulation.

Factor	Change
II	Unchanged
VII	Increased +++
VIII, IX, X, XII	Increased
XI	Reduced
Fibrinogen	Increased +++
Platelets	Stable

Gastrointestinal system:

Progesterone relaxes smooth muscle of the GIT consequently, it impairs esophageal and intestinal motility during pregnancy.

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PATHOPHYSIOLOGY OF PIH

Hypertension is the most common medical disorder of pregnancy affecting 6%-8% of pregnant women. It results in maternal and fetal complication such as preterm birth, intrauterine growth restriction, fetal and maternal death.

CLASSIFICATION OF HYPERTENSIVE DISORDERS:

Gestational hypertension:

In a woman with no preexisting hypertension or other signs or symptoms of preeclampsia, it manifests as elevated blood pressure after 20 weeks gestation that resolves by 12 weeks postpartum.

Preeclampsia:

It is defined as the new onset of hypertension and proteinuria after 20 weeks gestation. The National High Blood Pressure Education Program (NHBPEP) has recommended that clinicians consider the diagnosis of preeclampsia in the absence of proteinuria when any one of the following are present.

(1) Persistent epigastric or right upper quadrant pain (2) Persistent cerebral symptoms (3) fetal growth restriction (4) Thrombocytopenia and (5) Elevated serum liver enzymes.

Eclampsia:

The term eclampsia is used when CNS involvement results in the new onset of seizures in a woman with preeclampsia

HELLP syndrome:

The term HELLP syndrome refers to the development of Hemolysis, Elevated Liver enzymes, and Low Platelets in a women with preeclampsia.

Chronic hypertension:

It is defined as prepregnancy blood pressure levels ≥ 140 mmHg systolic or ≥ 90mmHg diastolic or elevated blood pressure that fails to resolve after delivery.

Diagnostic criteria for mild and severe preeclampsia

Mild preeclampsia	Severe preeclampsia
BP≥140/90mmHg after 20 weeks gestation Proteinuria(300mg/24 hr or 1+ result on dipstick specimen)	BP≥160/110 mmHg Proteinuria >5 gm/24 hr urine specimen (or≥3+ on two random urine samples at least 4 hours apart) Elevated serum creatinine Pulmonary edema Oliguria: urine output <500ml in 24 hrs Intrauterine growth restriction Headache Visual disturbances Epigastric or right upper quadrant

	pain due to stretching of Glisson's capsule by hepatic edema Impaired liver function Thrombocytopenia Signs of HELLP syndrome

Risk factors:

1. Women exposed to chorionic villi for the first time.
2. Women exposed to a superabundance of chorionic villi, as with twins or hydatidiform mole.
3. Have preexisting vascular disease, Obesity, Diabetes.
4. Women who are genetically predisposed to hypertension developing during pregnancy.

Prophylaxis and treatment of preeclampsia and eclampsia:

1) **Eclampsia:**

Pritchard regime: 20 ml of 20% MgSO4 slow I.V, followed by 20 ml of 50% $MgSO_4$ deep I.M, Thereafter 10 ml of 50% $MgSO_4$ 4^{th} hourly till 24 hours postpartum

Zuspan's regime: 4 gm $MgSo_4$ slow I.V followed by 1-2gm/hr I.V infusion

Sibai regime: 6 gm $MgSo_4$ slow I.V followed by 2 gm /hr I.V infusion

Lytic cocktail or Menon regime: 25 mg Chlorpromazine and 100 mg Pethidine I.V along with 50 mg Chlorpromazine and 25 mg Promethazine I.M. subsequently 50 mg Chlorpromazine and 25 mg Promethazine I.M. at 4 th hourly intervals for a period upto 24 hrs 100 mg Pethidine in 500 ml of 10% dextrose at 20 -30 drops/min.

Lean regime: Diazepam 40 mg I.V and further 40 mg in 500 ml of 5% dextrose at 30 drops/min

Phenytoin therapy: Initial dose of 10 mg/kg followed by 5mg/kg two hours later. Thereafter 200 mg orally after 12 hours continued until 48 hours.

A loading dose of 4-6gm Magnesium Sulphate diluted in 100ml of normal saline given over 15min intravenously. Then 2 gm/hr in 100ml of IV infusion. (maintain serum levels between 4 and 7mEq/L).

Intermittent injection:

4gm given slow IV followed by 10gm, 5gm in each buttocks as deep IM injection. Then every 4 hrs 5gm intramuscularly upto 24hrs after delivery.

PATHOGENESIS:

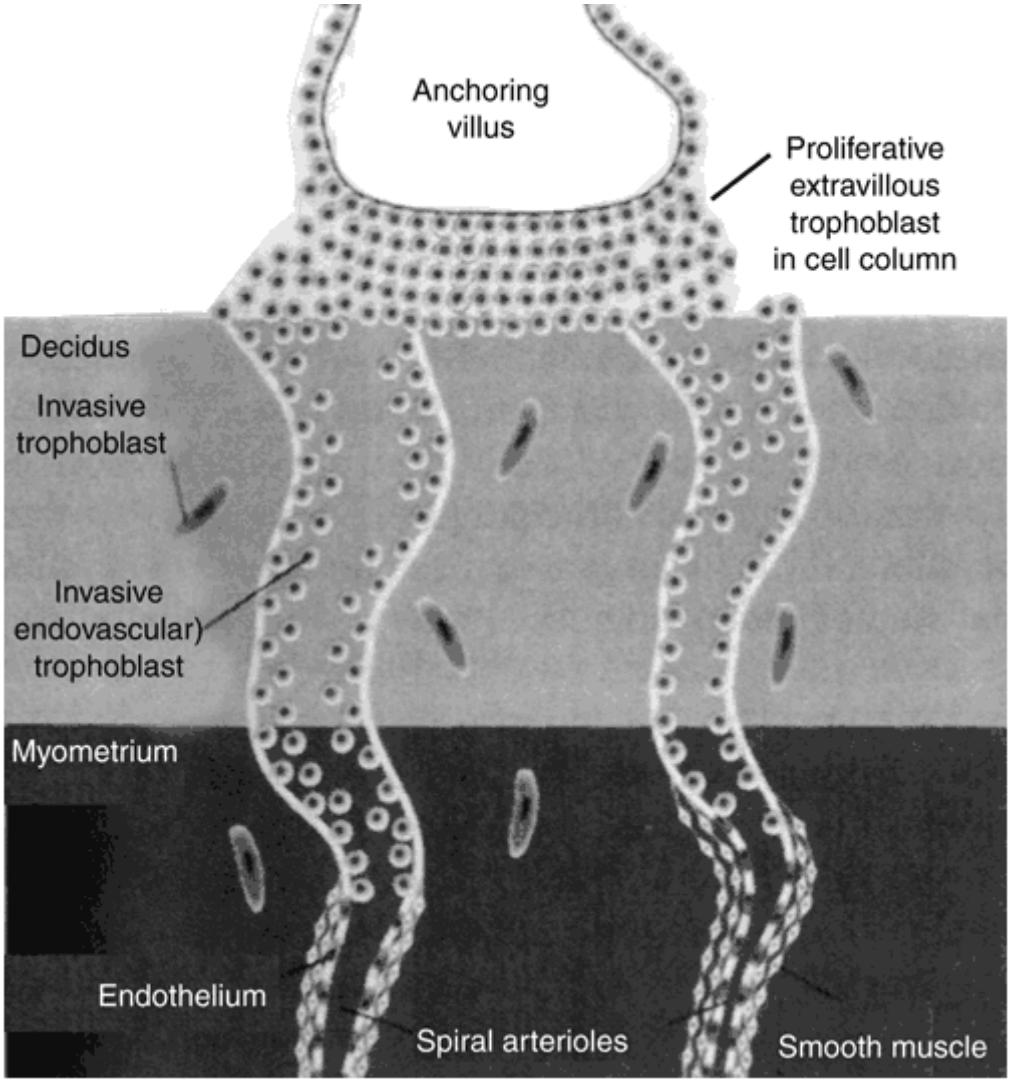
- Abnormal placentation and failure of normal angiogenesis and trophoblastic invasion resulting in hyper response to vasomotor stimuli.

- Widespread endothelial dysfunction and an accentuated systemic inflammatory response.

- Immunological intolerance between maternal and fetoplacental tissues and auto antibodies to angiotensin receptor-1

- Two endogenous antiangiogenic proteins namely Soluble fms-like tyrosine kinase-1 and Soluble endoglin are upregulated in preeclampsia.

- Abnormal vasospasm, imbalance of ThromboxaneA_2 and Prostaglandin$_{I2}$, altered handling of fatty acids by liver were other proposed pathogenesis of PIH.



CLINICAL MANIFESTATIONS:

Cardiovascular system:

- Severe vasospasm and greater sensitivity to catecholamines leads to hypertension.
- Severe preeclampsia is a Hyperdynamic state with increased cardiac output (CO), increased SVR.

Hematologic system:

- Reduction in blood volume causes a greater degree of hemoconcentration.
- Thrombocytopenia is most common hematologic abnormality
- Preeclampsia is relatively hypercoagulable state
- Altered relationship between plasminogen activators and inhibitors, with a resultant decrease in fibrinolytic activity, contributes to the persistence of fibrin in the renal and placental microvasculature. Preeclamptic women have higher concentrations of **lipoprotein (a),** which may be a marker for the severity of disease. Lipoprotein (a) enhances blood coagulation by competing with plasminogen for its binding sites on fibrin clots and endothelial cells.

Renal changes:

- Renal manifestations include persistent proteinuria, changes in GFR and Hyperuricemia
- **Oliguria** parallels the severity of disease, and persistent oliguria (less than 400 mL in 24 hours) requires assessment of volume status.

Endocrine:

- In women with **preeclampsia,** a breakdown of the normal balance between vasodilators (e.g., PGI_2, nitric oxide) and vasoconstrictors (e.g., angiotensin II, TXA_2, serotonin, endothelin) occurs.

There is also a decrease in plasma renin concentration and a suppression of the renin angiotensin-aldosterone system.

Respiratory system:

- Pharnygeal and laryngeal edema may make airway management difficult, necessitating the use of smaller endotracheal tubes.
- Decreased colloid osmotic pressure with greater vascular permeability increases the risk of pulmonary edema.

Hepatic changes:

- Serum transaminase levels frequently increase in patients with mild preeclampsia.
- Epigastric or sub costal pain is an ominous symptom that typically is caused by distention of the liver capsule by edema or subcapsular or parenchymal bleeding.

Neurologic changes

- The classic manifestations of preeclampsia include severe headache, visual disturbances, CNS hyper excitability, and hyperreflexia.
- The occurrence of seizures indicates eclampsia until proved otherwise.
- The etiology of eclamptic seizures remains unclear. Other proposed etiologies for eclamptic seizures include vasospasm, micro infarctions and punctate hemorrhages, thrombosis, and cerebral edema.

Uteroplacental perfusion:

- Uteroplacental perfusion is decreased in preeclampsia with the potential for intrauterine growth retardation

ANESTHETIC IMPLICATION:

1. Altered coagulation precludes regional anesthesia in severe preeclampsia

2. Exaggerated stress response during laryngoscopy, Hypotension on induction due to reduced intravascular volume

3. Difficult airway anticipated due to airway edema and capillary engorgement

4. Risk of aspiration due to delayed gastric emptying time

5. Acute pulmonary edema because of increased pulmonary capillary permeability

6. Strict hemodynamic control and monitoring, FHR monitoring is essential

7. Magnesium therapy potentiates non depolarising muscle relaxant

8. Consider perioperative seizure prophylaxis and pain relief

PHARMACOLOGICAL CHANGES IN PREGNANCY

PHARMACODYNAMICS OF LA

Pregnant women require small doses of local anesthetics due to epidural venous engorgement, enhance neural sensitivity to local anesthetics higher PH lower bicarbonate and total Carbondioxide content in CSF in women undergoing cesarian section.

PHARMACOKINETICS OF LA:

Bupivacaine is the most commonly used local anesthetic in obstetric anesthesia because it preserve motor function and is compatible with intrathecal opioids. It bound extensively by two proteins, both of which decline during pregnancy: (1) alpha-1-acid glycoprotein(AAG), a high-affinity, low-capacity site, and (2) albumin, a low-affinity, high-capacity site.

Preeclampsia and LA Drugs

In preeclampsia reduced hepatic blood flow, abnormal liver function and decreased intravascular volume affect, maternal blood concentration of local anesthetics. Long acting amides have a relatively low hepatic extraction ratio, changes in liver blood flow in preeclampsia may have less effect on the metabolic clearance

Effect on Uterus:

Pregnancy may enhance uterine vascular reactivity to local anesthetic agents

Effect on umbilical blood flow:

Bupivacaine does not constrict umbilical artery at clinically relevant concentration of 0.3-1 mcg/ml. At higher concentration the effect of bupivacaine appear to be biphasic. 5-10 mcg/ml produce uterine artery constriction more than 125 mcg/ml produce relaxation of artery.

S/D ratio (systolic peak to diastolic trough of the umbilical artery) in the umbilical artery decreases during normal pregnancy and high ratio usually are associated with fetal compromise.

Placental drug transfer:

Factor affecting placental transfer of drugs include

1. Physiochemical characteristic of local anesthetic agent
2. Concentration of free drug in maternal circulation
3. Permeability of the placenta
4. Hemodynamic events occurring within the fetal maternal unit

During pregnancy, anatomic adaptations result in substantial (near maximal) vasodilation of the uterine spiral arteries, this result in a low-resistance pathway for the delivery of blood to the placenta. Therefore, adequate uteroplacental blood flow depends on the maintenance of a normal maternal perfusion pressure.

Physical factors (e.g., molecular weight, lipid solubility, degree of ionization) affect the placental transfer of drugs and other substances. In addition, other factors affect maternal-fetal exchange, including changes in maternal and fetal blood flow, placental binding, placental metabolism, diffusion capacity, and degree of maternal and fetal plasma protein binding.

Lipophilicity, which enhances the central nervous system uptake of general anesthetic agents, also heightens the transfer of these drugs across the placenta. Fetal acidemia can result in the so-called “ion trapping” of both local anesthetics and opioids.

Molecular size:

Compound with a molecular size less than 1000 Daltons crosses the placenta easily.

Ionization and lipid solubility:

The degree of ionization affect the rate of placental diffusion because the unionized molecule is more lipid soluble than ionised molecule.

Protein binding:

Bupivacaine in the maternal plasma is 2 mg/L. bupivacaine are approximately 90% bound to maternal plasma proteins, the free concentration of drug available for placental transfer is 0.2 mg/L. At equilibrium, the concentration of free drug is equal on both sides of the placenta. However, in the fetus, bupivacaine 50% bound to fetal plasma proteins, Total bupivacaine, the concentration in fetal plasma is 0.4 mg/L and an F/M ratio of 0.2.

Transfer across the placenta may be reported as drug clearance or as a ratio that is also referred to as the *transfer index* used to improve interplacental comparisons.

Teratogenicity:

Local anesthetics used during the first trimester of pregnancy caused reversible reduction of cell division in tissue culture. Large multicenter study demonstrated that the risk of congenital anomalies in humans was not increased by the administration of benzocaine, procaine, tetracaine, or lidocaine during early pregnancy. However, a twofold increase in the incidence of congenital anomalies was noted in infants whose mothers had received mepivacaine.

FETUS AND NEWBORN:

Pharmacokinetics:

Local anesthetics, once transferred across the placenta, are distributed in the fetus. Factors that influence tissue uptake of the drug include (1) fetal plasma protein binding, (2) lipid solubility, (3) the degree of ionization of the

drug, and (4) hemodynamic changes that affect the distribution of fetal cardiac output.

The term newborn has the hepatic enzymes necessary for the biotransformation of amide local anesthetics. The elimination half-life of these drugs is longer in the neonate compared with the adult. The use of mepivacaine in obstetric epidural analgesia elimination half-life of the drug in the newborn was approximately 9 hours.

SYSTEMIC TOXICITY:

Changes in fetal heart rate (FHR) after administration of local anesthetics most often are related to indirect effects such as maternal hypotension and uterine hyperstimulation.FHR patterns are not affected by the larger doses of local anesthetics required during administration of epidural anesthesia for cesarean delivery

PRETERM FETUS AND NEWBORN:

Enhanced drug sensitivity in the preterm newborn: (1) less protein is available for drug binding; (2) higher levels of bilirubin are present and may compete with the drug for protein binding; (3) greater drug access to the CNS occurs because of a poorly developed blood-brain barrier; (4) the preterm infant has greater total body water and less fat content; and (5) the preterm infant has a decreased ability to metabolize and excrete drugs

The placenta efficiently eliminates fetal bilirubin. Thus the hyperbilirubinemia of prematurity normally occurs in the postpartum period. Bupivacaine has been implicated as a possible cause of neonatal jaundice. High affinity of the drug for fetal erythrocyte membranes may lead to a decrease in filterability and deformability, which may render red blood cells more prone to hemolysis.

Asphyxia:

In asphyxiated preterm fetus , exposure to bupivacaine reduced blood flow to vital organs however, fetal heart rate, blood pressure, and acid-base measurements did not change Johnson et al. suggested that bupivacaine might be preferable to lidocaine in the presence of fetal acidosis because the greater maternal protein binding of bupivacaine may limit its placental transfer

Pharmacokinetic principles

Transfer of a drug that is highly protein bound is affected by the concentration of both maternal and fetal plasma proteins. The pKa of a drug determines the fraction of drug that is nonionized at physiologic pH. Thus, fetal acidemia will greatly enhance the maternal-to-fetal transfer (i.e., “ion trapping”) of many *basic* drugs, such as local anesthetics and opioids.

Factor affecting placental transfer of drug (maternal to fetal)

	Increased transfer	**Decreased transfer**
Size-Mol. Weight(Dalton)	<1000	>1000
Charge of molecule	Uncharged	Charged
Lipid solubility	Lipophilic	Hydrophilic
PH vs drug Pka	Higher proportion of un-ionised drug in maternal plasma	Higher proportion of ionised drug in maternal plasma
Placental efflux transporter proteins(e.g.P-glycoprotein)	Absent	Present
Binding protein type	Albumin (lower binding affinity)	Alpha-1-acid glycoprotein(AAG) higher binding affinity
Free (unbound)drug fraction	High	Low

Transplacental transfer of anesthetic drug

Opioids:

Opioids have long been the mainstay for systemic pain relief in obstetric patients. Safety associated with their use has increased because of a better understanding of their pharmacokinetics, improved monitoring, and the ability to reverse adverse effects with the antagonist naloxone.

Fentanyl and its analogs currently are used extensively as intrapartum analgesics. These drugs are administered by the epidural, intrathecal, and intravenous routes. Fentanyl has a high degree of lipophilicity and albumin

binding (84%). Maternal epidural administration of fentanyl results in an F/M ratio between 0.37 and 0.57. During early pregnancy, fentanyl is rapidly transferred and may be detected not only in the placenta but also in the fetal brain.

Opioid transfer during In Vitro perfusion of the Human Placenta

	Morphine	**Meperidine**	**Alfentanil**	**Fentanyl**	**Sufentanil**
Lipid solubility	1.4	39	129	816	1727
Percent nonionized at pH 7.4	23%	7.4%	89%	8.5%	20%
Percent protein binding	30%	70%	93%	84%	93%
Placenta drug ratio	0.1	0.7	0.53	3.4	7.2
F/M ratio, MTF	0.08	0.27	0.22	0.19	0.14
F/M ratio, FTM	0.08	0.13	0.11	0.08	0.18
Minutes to steady state	30	20	20	40-60	40-60
Clearance index MTF	0.4	0.95	0.75	0.76	0.41
Clearance index FTM	0.5	0.91	0.78	0.61	0.76

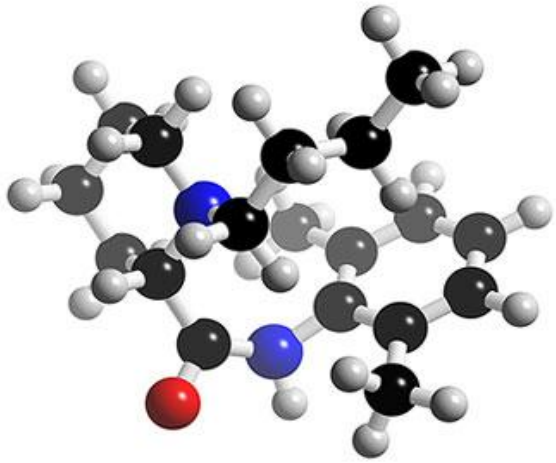
ANESTHETIC DOSE REQUIREMENT:

The effects of pregnancy on local anesthetic potency may reflect a combined effect of mechanical factors associated with pregnancy (i.e., dilated epidural veins decrease the volume of the epidural and subarachnoid spaces) and direct effects of hormones, especially progesterone, on the susceptibility of nerves to conduction blockade by local anesthetics per se. Hormonal alterations are probably the more important of these two factors because greater spread of epidural anesthesia occurs during the first trimester of pregnancy, before any gross change in vascular dimensions within the epidural or subarachnoid spaces. The dosage of local anesthetics should probably be reduced in patients in all stages of pregnancy.

Pregnancy enhances the spread of hyperbaric local anesthetic solution in the subarachnoid space, resulting in a 25% reduction in the segmental dose requirement (i.e., milligrams of drug necessary to block one spinal segment) in term pregnant women.

PHARMACOLOGY OF BUPIVACAINE

Structure



It is an amide local anaesthetic first synthesized in Sweden by **Ekenstam** and his colleagues in 1957 and used clinically by **L.J.Telivuo** in 1963. Its molecular weight is 288.

PHARMACOKINETIC

At pH 7.4 only 15% exists in nonionised form. Absorption depends on the site of injection, dosage and use of epinephrine. Primarily metabolized by the liver and Lung is capable of extracting bupivacaine from circulation.

Pka	: 8.1
Protein Binding	: 95%
Lipid solubility	: 28
Volume of distribution	: 73 litre

Clearance of drug from plasma : 0.471 lit/min

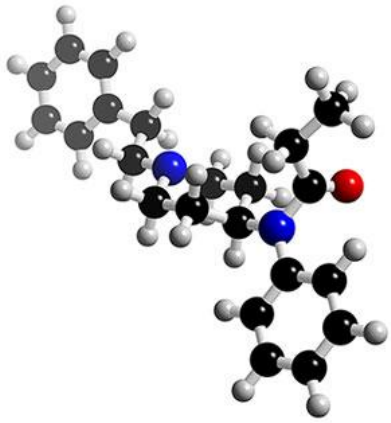
Elimination half life : 210 min (3.5 hours)

Onset time : 5 -7 min

F/M ratio : 0.2-0.4

PHARMACOLOGY OF FENTANYL

Structure



Fentanyl is a synthetic phenylpiperidine opioid, of the 4-anilopiperidine

PHYSICOCHEMICAL PROFILE

Molecular weight : 528. 29

PKa : 8.4

% unionized at pH 7.4 :8.5 %

% bound to plasma proteins : 84 %

Potency : 80 >Morphine

F/M ratio, MTF : 0.19

F/M ratio, FTM : 0.08

Placenta drug ratio : 3.4

Clearance index, MTF : 0.76

Clearance index, FTM : 0.61

Pharmacokinetic Profile

Volume of distribution at steady state(VDss) : 335litres

Clearance : 1530 ml/min

Effect- site equilibration time : 6.8 min

Hepatic extraction ratio : 0.8-0.1

Context – sensitive half time (4 hrs infusion) : 260 mins

Elimination half time : 3.1 to 6.6 hours

First pass pulmonary uptake : 75%

Neuraxial administration

The placement of opioids in the subarachnoid space to that opioid receptors (principally μ receptors) are present in the substantia gelatinosa of the spinal cord. Analgesia produced by neuraxial opioids, in contrast to the intravenous administration of opioids or regional anesthesia with local anesthetics, is not associated with sympathetic nervous system denervation,

skeletal muscle weakness, or loss of proprioception. Analgesia is dose related (epidural dose is 5 to 10 times the subarachnoid dose) and specific for visceral rather than somatic pain

Pharmacokinetics:

Fentanyl has the same baricity as cerebrospinal fluid at room temperature and addition to hyperbaric lignocaine or bupivacaine makes the solution hyperbaric. On injection into subarachnoid space, fentanyl mixes with CSF and attaches itself to spinal opioid receptors. Protein binding of drug in the CSF is negligible and the concentration of opioid in the CSF is thus free drug concentration. CSF dynamics do not provide any means of drug removal. Diffusion into the spinal cord and absorption into the blood flowing through spinal cord must remove all the fentanyl. This rate determining step of drug removal is likely to be the rate constant for fentanyl transfer from CSF to spinal cord and this rate constant is directly related to lipophilicity. Fentanyl can also migrate from the CSF into epidural vascular compartment via the duramater. However details of systemic pharmacokinetics of fentanyl are not known. Once in the CSF. fentanyl like other opioids, spreads rostrally. Because of the high affinity of fentanyl with binding sites in the lipid-rich Spinal cord, only 10% of administered dose migrates to cervical region.

Application

Intrathecal fentanyl is usually combined with local anaesthetics for Perioperative anesthesia and analgesia particularly in obstetrics. Fentanyl

administration intrathecally provides more intense and complete analgesia at rest, at a lower dose requirement when compared to the epidural or intravenous routes.

Side effects of intrathecal fentanyl

The four classic side effects of neuraxial opioids are pruritus, nausea and vomiting, urinary retention, and depression of ventilation.

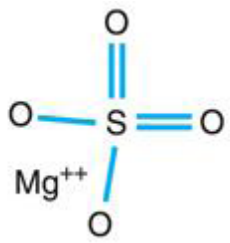
Pruritus is the most common side effect (often localized to face, neck, upper thorax) of neuraxial opioids. Pruritus induced by neuraxial opioids is not due to histamine release but is likely due to the cephalad migration of the opioid in CSF and its subsequent interaction with opioid receptors in the trigeminal nucleus.

Depression of Ventilation

The most serious side effect of neuraxial opioids is depression of ventilation, which may occur within minutes of administration or may be delayed for hours. The incidence of ventilatory depression requiring intervention after conventional doses of neuraxial opioids is about 1%,

MAGNESIUM

Chemical structure of Magnesium



Magnesium was discovered in 1755 by Sir Humpry Davy. Magnesium is the fourth plentiful cation in the body and second most abundant intracellular cation after potassium. It is a cofactor in hundreds of enzymatic reactions. It is a natural calcium antagonist.

Normal physiology:

Magnesium is a bivalent cation with atomic weight of 24.312. Human body contains one mole (24 gms) of magnesium of which 60% is present in bones, 20% is in muscles, 20% is in soft tissues. Only 1% of total body magnesium is present extracellularly.

Intracellular magnesium exists largely (90%) in bound form in ATP molecules of cytoskeleton (nucleus, mitochondria & reticulum). Only a small portion (5 – 14%) remains as ionized form within the cell.

Properties of Magnesium:

1. Magnesium intervenes in the activation of Ca ATPase & Na- K ATPase involved in transmembrane ion exchange. It acts as a stabilizer of cell membrane and intracytoplasmic charges.

2. Magnesium has antagonist action on L type calcium channels. It inhibits Ca inflows into the cell and outflow of Ca from sarcoplasmic reticulum.

3. Magnesium has antagonist effect on N-methyl D-aspartate receptors in nervous system.

4. Intracellular ionized magnesium is involved in phosphorylation & is necessary for activation of hundreds of enzymatic reactions concerning ATP.

Normal serum concentration:

1.6 to 2.6 mEq /L

Pharmacological Effects:

Cardiovascular system: Magnesium causes vasodilatation and may cause hypotension at high doses. It slows the rate of SA node impulse formation and prolongs SA node conduction time, the PR interval and AV nodal effective refractory period. Magnesium attenuates both vasoconstrictor and arrythmogenic actions of adrenaline.

Respiratory system: Magnesium is an effective bronchodilator and attenuates hypoxic pulmonary vasoconstriction.

Central nervous system: Magnesium is a CNS depressant and exhibits anticonvulsant properties. High concentrations inhibit catecholamine release from adrenergic nerve terminals and adrenal medulla.

Gastrointestinal system: Magnesium sulphate acts as a antacid when administered orally.

Genito urinary system: Magnesium has renal vasodilator and diuretic effect.

Toxicity / side effects:

Minor side effects include warmth, flushing, nausea, headache and dizziness. Dose related side effects include somnolence, areflexia, AV and intraventricular conduction disorders, progressive muscle weakness, and cardiac arrest.

USES:

1) Magnesium sulphate has a tocolytic effect at serum levels of 8-10mEq/L.

Loading dose of 4-6gm over 20min intravenously, then after the contraction ceases maintenance is done using 2-4gm per hour intravenously for 12-24 hours.

2) Magnesium sulphate is used in the dose of 50 mg intrathecally for potentiation of opioid analgesia.

3) To reduce the stress response during intubation, magnesium sulphate is used in the dosage of 30-50mg/kg. intravenously.

4) Hypomagnesemia: in case of mild deficiency 1gm every 6 hours for 4 doses, in severe cases 1-5gms (2 – 10ml of 50% solution) in divided doses, repeated until the serum levels are normal.

PRECAUTIONS:

During $MgSO_4$ therapy monitor-urine output, respiratory rate, knee jerk.

Effect of increasing plasma magnesium level

Plasma magnesium (MEq/L)	Effects
1.5-2.0	Normal plasma level
4.0-8.0	Therapeutic range
5.0-10	Electrocardiographic changes(PQ interval prolonged, QRS complex widens)
10-12	Loss of deep tendon reflexes
15-20	Sinoatrial and atrioventricular block, Respiratory paralysis
25	Cardiac arrest

REVIEW OF LITERATURE

1) **Comparison of intrathecal magnesium, fentanyl, or placebo combined with bupivacaine 0.5% for parturients undergoing elective cesarean delivery** Unlugenc and Ozalevli et al in march 2009 conducted a prospective, randomized, double-blind study to investigate the sensory, motor, and analgesic block characteristics of intrathecal magnesium 50 mg compared with fentanyl 25 μg and saline when added to 0.5% bupivacaine. METHOD : 90 ASA I and II healthy parturients undergoing elective cesarean section were included in the study. Onset and duration of sensory and motor block, maximal sensory block height, the time to reach the maximal dermatomal level of sensory block, and the duration of spinal anesthesia were recorded. CONCLUSION : They concluded that in patients undergoing cesarean section with spinal anesthesia, the addition of magnesium sulfate (50 mg) intrathecally to 10 mg of spinal bupivacaine (0.5%) did not shorten the onset time of sensory and motor blockade or prolong the duration of spinal anesthesia, as seen with fentanyl. (Acta Anes Scand 2009: 53 : 346-353)

2) **Combined intrathecal and epidural magnesium sulphate supplementation of spinal anesthesia to reduce post-operative analgesia requirements** R.Arcioni et al in 2007 conducted a prospective , randomized, double blind, controlled study in 120 ASA I and II patients coming for lower limb orthopedic surgery. METHOD :

Patients were randomly divide into 4 groups assigned to receive intrathecal magnesium sulphate, epidural magnesium sulphate, combined intrathecal and epidural magnesium sulphate or spinal anesthesia alone. Post-operative morphine consumption was assessed in all patients using PCA.

CONCLUSION : In patients undergoing orthopedic surgery, supplementation of spinal anesthesia with combined intrathecal and epidural magnesium significantly reduces the post-operative analgesic requirements. (Acta Anes Scand. 2007 ; 51(4): 482-489).

3) **Intrathecal Magnesium Prolongs Fentanyl Analgesia Ashokumar Buvanendran et al in 2002 were one of the first to administer magnesium intrathecally in humans. They conducted a prospective randomized controlled trial in 52 healthy parturient mothers requiring labour analgesia.**

METHOD: Patients were randomized to receive either intrathecal fentanyl 25 μg plus saline or fentanyl 25 μg plus magnesium sulfate 50 mg as part of a combined spinal-epidural technique. The duration of analgesia of the intrathecal drug combination was defined by the time of patient request for additional analgesia. RESULT: There was significant prolongation in the median duration of analgesia in the magnesium plus fentanyl group compared with the fentanyl alone group.

CONCLUSION: They concluded that intrathecal magnesium prolongs spinal opioid analgesia in humans and suggest that the availability of an intrathecal *N*methyl-D-aspartate antagonist could be of clinical importance for pain management.

(Anesth Analg 2002;95:661-666).

4) **Intrathecal versus intravenous fentanyl for supplementation of subarachnoid block during cesarean delivery** Sahar and Marie et al in 2002 conducted a randomized study in 48 healthy parturient patients undergoing elective cesarean section. One group received 12 mg of hyperbaric bupivacaine plus 12.5 μg of fentanyl intrathecally. Another group received 12 mg of hyperbaric bupivacaine intrathecally and 12.5 μg of fentanyl intravenously immediately after spinal. They found that additional intravenous fentanyl was needed in IV fentanyl group and incidence of hypotension and use of ephedrine was more in IV fentanyl group. The time to the first request for postoperative analgesia was significantly longer in the intrathecal fentanyl group than in the IV fentanyl group. They concluded that supplementation of spinal bupivacaine anesthesia for cesarean delivery with intrathecal fentanyl provides a better quality of anesthesia and is associated with a decreased incidence of side effects as compared with supplementation with the same dose of IV fentanyl (Anesth Analg 2002;95:209-213).

5) **The effect of adding intrathecal magnesium sulphate to morphine for postoperative analgesia after caesarean section** Khemakhem K et

al in 2006 conducted a prospective, randomized, double blind, controlled study to know the effect of intrathecal magnesium sulphate, morphine and their association in postoperative analgesia. METHOD: 97 ASA I and II parturients undergoing cesarean section are randomly allocated to 3 groups to receive 0.1 mg of morphine or 100 mg of magnesium or both.

Postoperative analgesia with the visual analogic score (VAS), analgesic requirement, and side effects were recorded. CONCLUSION: The addition of intrathecal magnesium sulphate 100 mg to morphine improved quality and duration of the postoperative analgesia with a better maternal satisfaction without additional side effects. (Euro Anes 2006 jun; 23: 183-4).

6) **Magnesium infusion reduces perioperative pain** Kara H, Sahin N, Ulusan V, Aydogdu T in 2002 conducted a study to determine whether perioperative infusion of magnesium would reduce postoperative pain and anxiety. METHOD: Twenty-four patients, undergoing elective hysterectomy, received a bolus of 30 mg/kg magnesium sulphate or the same volume of isotonic sodium chloride solution intravenously before the start of surgery and 0.5 g/hr infusion for the next 20 hr. Intraoperative and postoperative analgesia were achieved with fentanyl and morphine respectively. Patients were evaluated pre- and postoperatively for anxiety. CONCLUSION: Continuous magnesium infusion, including the pre-, intra-, and postoperative periods reduces

analgesic requirements and reduces the anxiety of the patients. (Eur J Anesthesiol. 2002 Jan;19(1):52-6).

7) **Role of magnesium sulfate in postoperative analgesia** Tramer MR et al in 1996 conducted one of the earliest studies to demonstrate the anti nociceptive characteristics of magnesium. METHOD In a randomized double – blind study, they included 42 ASA I and II patients undergoing abdominal hysterectomy. Study group received 15 ml of 20% magnesium before start of surgery and an infusion of 2.5 ml/hr for next 20 hrs. Control group received same amount of normal saline. Maximum expiratory flow (peak flow), pain at rest and during peak flow and discomfort were evaluated up to the 48th postoperative hour and 1 week and 1 month after surgery. Insomnia was evaluated after the first and second postoperative nights. CONCLUSION: They concluded that the perioperative application of magnesium sulphate is associated with smaller analgesic requirement, less discomfort, and a better quality of sleep in the postoperative period but not with adverse effects. (Anaesthesiology;1996:84(2) 340-7).

MATERIALS AND METHODS

Patient selection:

After obtaining ethical committee approval from Govt. Kilpauk Medical College. 60 Pregnant women with mild PIH undergoing elective Caesarean section ASA I and II between the age group of 18-35 under spinal anesthesia were randomly divided into three groups.

Minimal fasting period is 8hrs, IV line secured with 18G venflon All patients received premedication with Inj. Ranitidine 50mg IV and Inj. Metoclopramide 10 mg IV, 10 min before surgery and preloaded with RL 10-12ml /kg. All patients received 5L of O_2 / min through mask throughout procedure Patients were treated with titrated doses of

- Inj. Ephedrine 6mg I.V if systolic BP<90mmhg
- Inj. Atropine 0.6mg I.V if heartrate <60/min

After delivery of baby Inj. Oxytocin 10 IU in drip and 10 IU IM given

INCLUSION CRITERIA	EXCLUSION CRITERIA
ASA I and II	Contraindications to spinal anesthesia
Age between 18-35 years	Heart disease
Mild PIH (BP<160/110mmHg)	Fetal distress
Elective Caesarean Section	Eclampsia
	Allergy to local anesthetic drugs
	Seizure disorders
	Patient with coagulation disorders

GROUPS:

Group C

Control group, (N=20) patients 0.5% 2cc (10mg) Bupivacaine + 0.6cc normal saline.

Group F:

Fentanyl (N= 20) patients received 0.5% 2cc Bupivacaine +0.5cc (25mic gm) Fentanyl +0.1cc NS.

Group M:

$MgSO_4$ group (N=20), 0.5% 2cc Bupivacaine +0.5cc Fentanyl +0.1cc 50%(50mg) $MgSO_4$.

All patients were monitored with ECG, NIBP, Pulse Oximetry. Respiratory rate, urinary output and knee jerk were also monitored. Under aseptic precaution patient in right lateral decubitous position by mid line approach spinal anesthesia was performed according to the study groups. Wedge was placed to prevent decreasing venous return due to aortocaval compression.

The local anesthetic drug prepared by the assistant according to the group and given to his performer who injected drug by spinal anesthesia without knowing the content of the drug he records his findings needed for the study.

Observation

The onset of sensory blockade, motor blockade, upper level of analgesia, intensity of motor block, two segment regression time, APGAR Score, Postoperative analgesia duration and hemodynamic parameters at 1,5,10,15,20,30 minutes were observed.

Motor block was assessed by Bromage motor score and sedation by Ramsay sedation score.

Sensory score:

Score	response
0	normal sensation
1	analgesia (loss of pin prick sensation)
2	anesthesia (loss of touch sensation)

Bromage motor score

Grade	Response	Degree of block
0	no motor block	nil(0%)
1	unable to straight leg raise	partial(33%)
2	unable to flex knee against resistance	almost complete(66%)
3	unable to flex ankle	complete

Ramsay sedation Score:

Score	Response
1	anxious or restless or both
2	Co-operative, oriented & tranquil
3	responds to commands
4	brisk response to stimulus
5	sluggish response to stimulus
6	no response to stimulus

SENSORY BLOCK ONSET TIME

Time interval between end of anesthetic injection and appearance of cutaneous analgesia in dermatomes T-12,T-10,T-8,T-6

DURATION OF MOTOR BLOCK

Administration of anesthetic and attainment of grade 0 in Bromage motor scale

DURATION OF ANALGESIA

Administration of anesthetic and disappearance of cutaneous level of sensation at each dermatomal level

POST-OP ANALGESIA DURATION

Administration of anesthetic and time of analgesic requirement in PACU.

OBSERVATION AND RESULTS

Descriptives

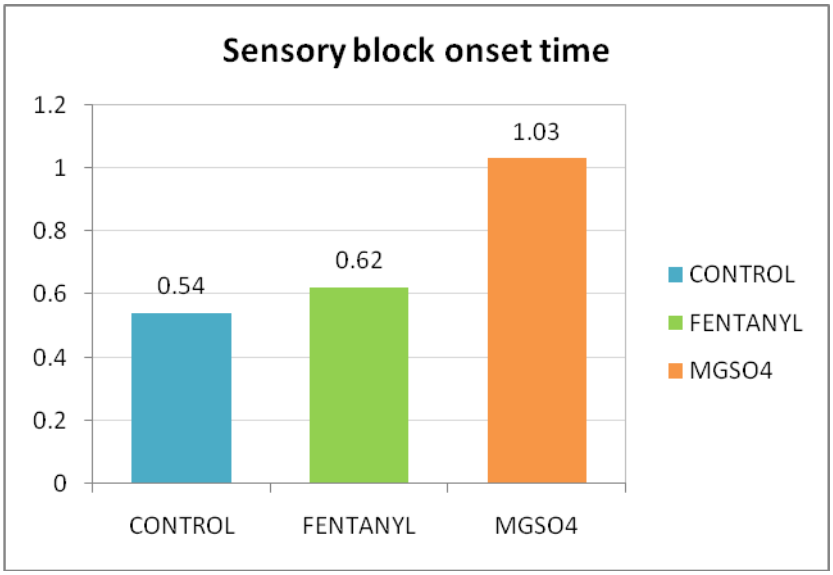
Sensory block onset time

	N	Mean	Std. Deviatic	Std. Erro	95% Confidence Interva Mean		Minimun	Maximun
					Lower Boun	Upper Boun		
FENTANYL	20	.62	.204	.046	.52	.72	1	1
CONTROL	20	.54	.123	.028	.49	.60	1	1
$MgSO_4$	20	1.03	.424	.097	.82	1.23	1	2
Total	60	.73	.345	.045	.64	.82	1	2

ANOVA

Sensory block onset time

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	2.594	2	1.297	16.816	.000
Within Groups	4.318	56	.077		
Total	6.912	58			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

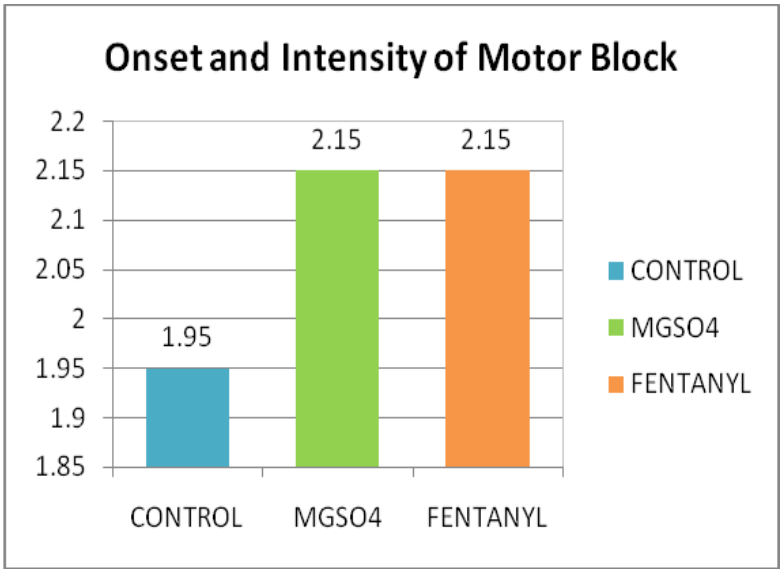
Onset and intensity of motor block

	N	Mean	Std. Deviatio	Std. Erro	95% Confidence Interval Mean		Minimum	Maximum
					Lower Boun	Upper Boun		
FENTANYL	20	2.15	.489	.109	1.92	2.38	1	3
CONTROL	20	1.95	.394	.088	1.77	2.13	1	3
$MgSO_4$	20	2.15	.489	.109	1.92	2.38	1	3
Total	60	2.08	.462	.060	1.96	2.20	1	3

ANOVA

Onset and intensity of motor block

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	.533	2	.267	1.261	.291
Within Groups	12.050	57	.211		
Total	12.583	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

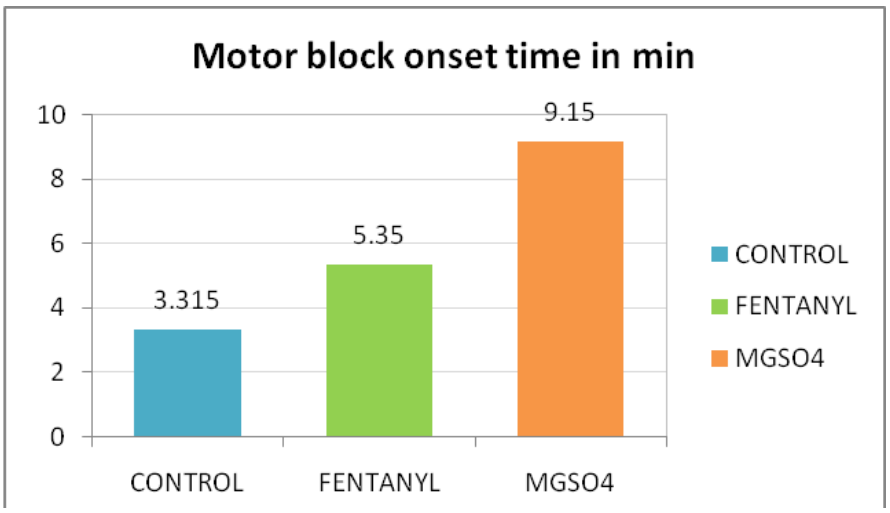
Motor block onset time in min

	N	Mean	Std. Deviat	Std. Erro	95% Confidence Inter Mean		Minimur	Maximu
					Lower Bou	Upper Bou		
FENTANYL	20	5.350	1.3387	.2993	4.723	5.977	3.0	8.0
CONTROL	20	3.315	.8707	.1947	2.907	3.723	2.0	5.0
$MgSO_4$	20	9.150	1.623(	.3629	8.390	9.910	6.5	12.0
Total	60	5.938	2.7598	.3563	5.225	6.651	2.0	12.0

ANOVA

Motor block onset time in min

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	350.856	2	175.428	101.511	.000
Within Groups	98.506	57	1.728		
Total	449.362	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

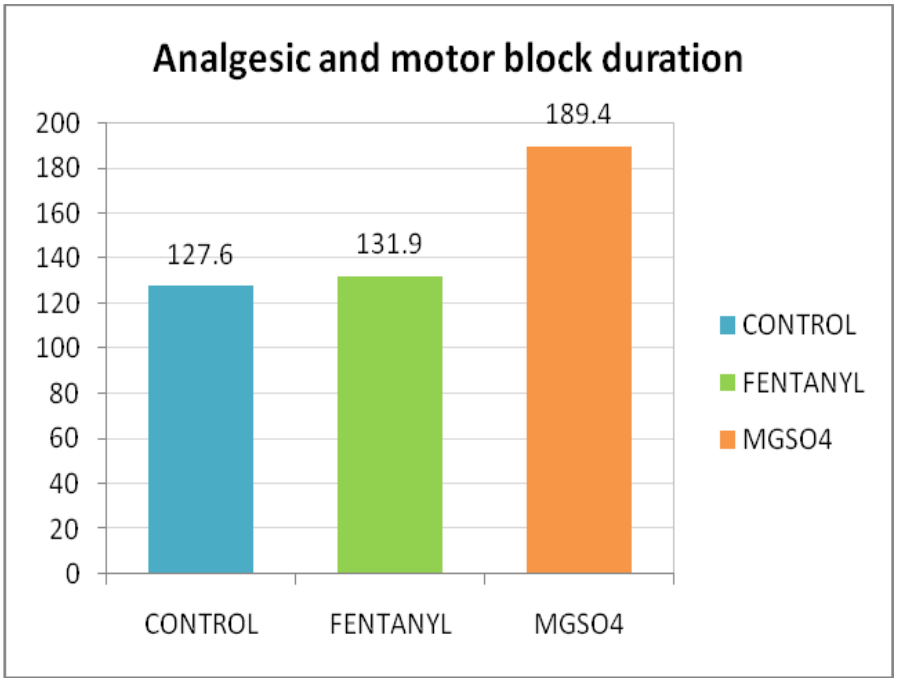
Analgesic and motor block duration in min

	N	Mean	Std. Deviati	Std. Erro	95% Confidence Interv Mean		Minimur	Maximur
					Lower Bour	Upper Bour		
FENTANYL	20	131.90	11.634	2.602	126.45	137.35	110	156
CONTROL	20	127.60	14.065	3.145	121.02	134.18	100	145
$MgSO_4$	20	189.40	19.329	4.322	180.35	198.45	156	218
Total	60	149.63	32.169	4.153	141.32	157.94	100	218

ANOVA

Analgesic and motor block duration in min

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	47626.533	2	23813.267	101.073	.000
Within Groups	13429.400	57	235.604		
Total	61055.933	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

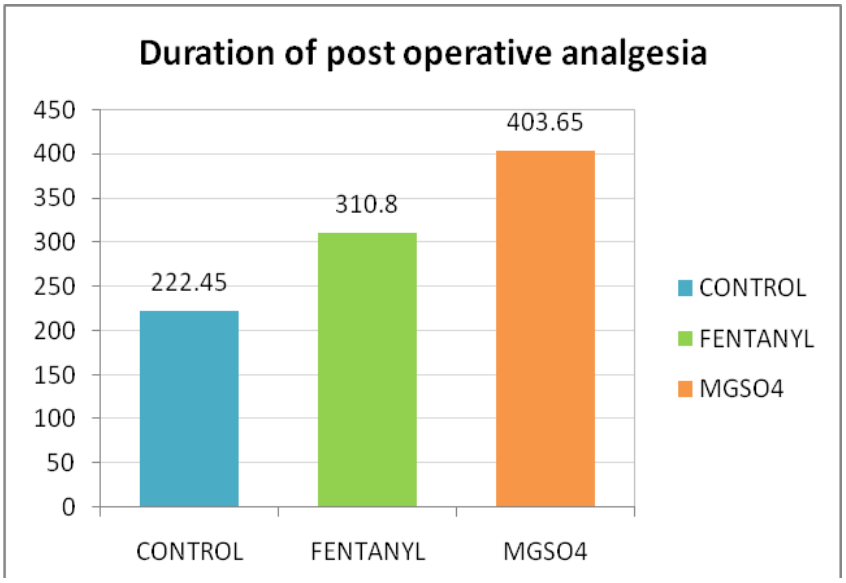
Duration of post operative analgesia in min

	N	Mean	Std. Deviati	Std. Erro	95% Confidence Interv Mean		Minimur	Maximur
					Lower Bour	Upper Bou		
FENTANYL	20	310.80	25.548	5.713	298.84	322.76	262	338
CONTROL	20	222.45	13.942	3.117	215.93	228.97	198	242
$MgSO_4$	20	403.65	27.186	6.079	390.93	416.37	371	460
Total	60	312.30	77.955	10.064	292.16	332.44	198	460

ANOVA

Duration of post operative analgesia in min

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	328401.900	2	164200.950	310.567	.000
Within Groups	30136.700	57	528.714		
Total	358538.600	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

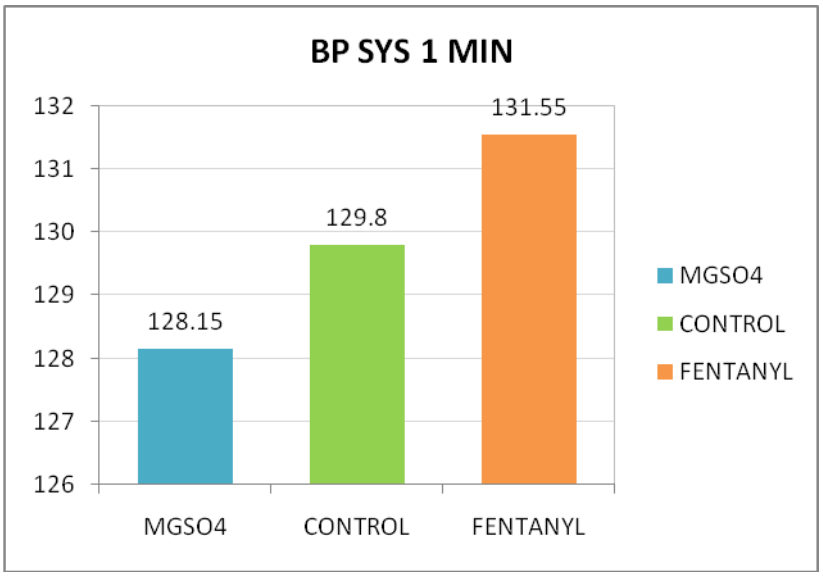
BP SYS 1 MIN

	N	Mean	Std. Deviatio	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
FENTANYL	20	131.55	6.108	1.366	128.69	134.41	122	142
CONTROL	20	129.80	17.698	3.957	121.52	138.08	90	152
$MgSO_4$	20	128.15	14.431	3.227	121.40	134.90	98	154
Total	60	129.83	13.487	1.741	126.35	133.32	90	154

ANOVA

BP SYS 1 MIN

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	115.633	2	57.817	.310	.734
Within Groups	10616.700	57	186.258		
Total	10732.333	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

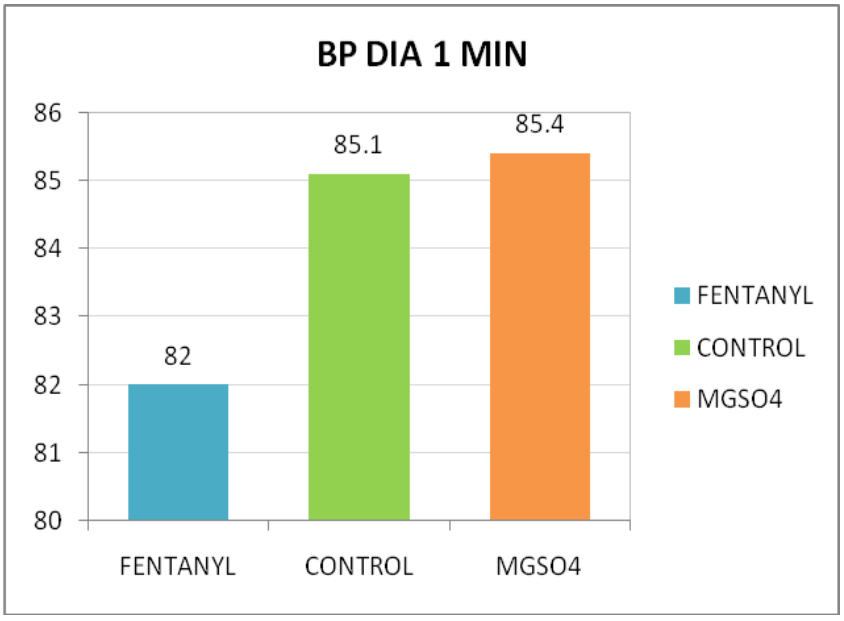
BP DIA 1 MIN

	N	Mean	Std. Deviat	Std. Err	95% Confidence Inter for Mean		Minimu	Maximu
					Lower Bou	Upper Bou		
FENTANYL	20	82.00	7.398	1.654	78.54	85.46	66	92
CONTROL	20	85.10	13.082	2.925	78.98	91.22	60	108
$MgSO_4$	20	85.40	10.763	2.407	80.36	90.44	60	100
Total	60	84.17	10.604	1.369	81.43	86.91	60	108

ANOVA

BP DIA 1 MIN

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	141.733	2	70.867	.622	.540
Within Groups	6492.600	57	113.905		
Total	6634.333	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

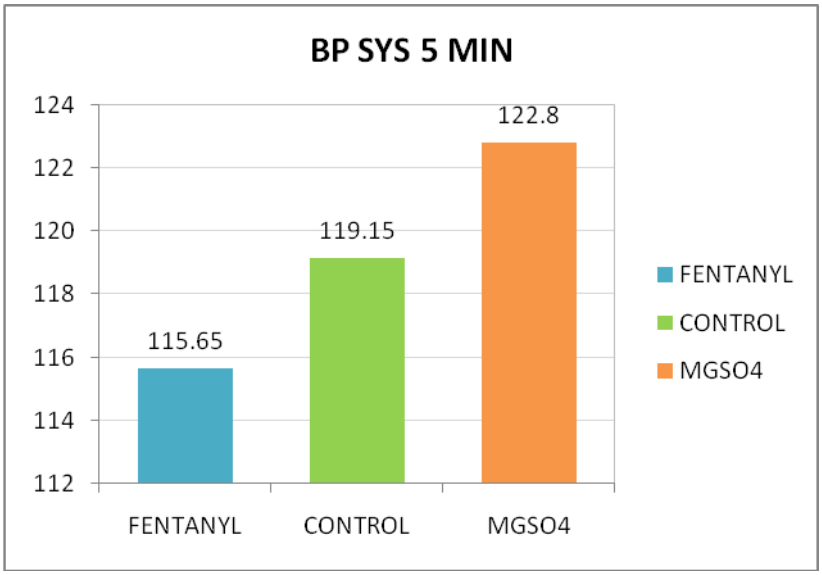
BP SYS 5 MIN

	N	Mean	Std. Deviati	Std. Erro	95% Confidence Interv for Mean		Minimur	Maximur
					Lower Bour	Upper Bour		
FENTANYL	20	115.65	13.647	3.052	109.26	122.04	90	146
CONTROL	20	119.15	14.376	3.215	112.42	125.88	80	142
$MgSO_4$	20	122.80	10.476	2.343	117.90	127.70	100	146
Total	60	119.20	13.059	1.686	115.83	122.57	80	146

ANOVA

BP SYS 5 MIN

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	511.300	2	255.650	1.526	.226
Within Groups	9550.300	57	167.549		
Total	10061.600	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

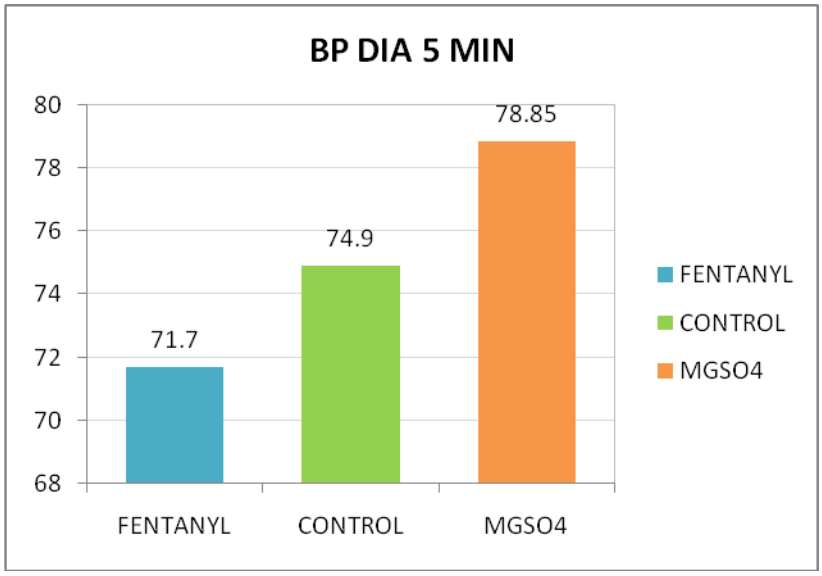
BP DIA 5 MIN

	N	Mean	Std. Deviatio	Std. Erro	95% Confidence Interval Mean		Minimum	Maximum
					Lower Boun	Upper Boun		
FENTANYL	20	71.70	9.804	2.192	67.11	76.29	52	92
CONTROL	20	74.90	8.955	2.002	70.71	79.09	58	90
$MgSO_4$	20	78.85	9.115	2.038	74.58	83.12	60	96
Total	60	75.15	9.604	1.240	72.67	77.63	52	96

ANOVA

BP DIA 5 MIN

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	513.100	2	256.550	2.967	.059
Within Groups	4928.550	57	86.466		
Total	5441.650	59			



Ephedrine requirement:

Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

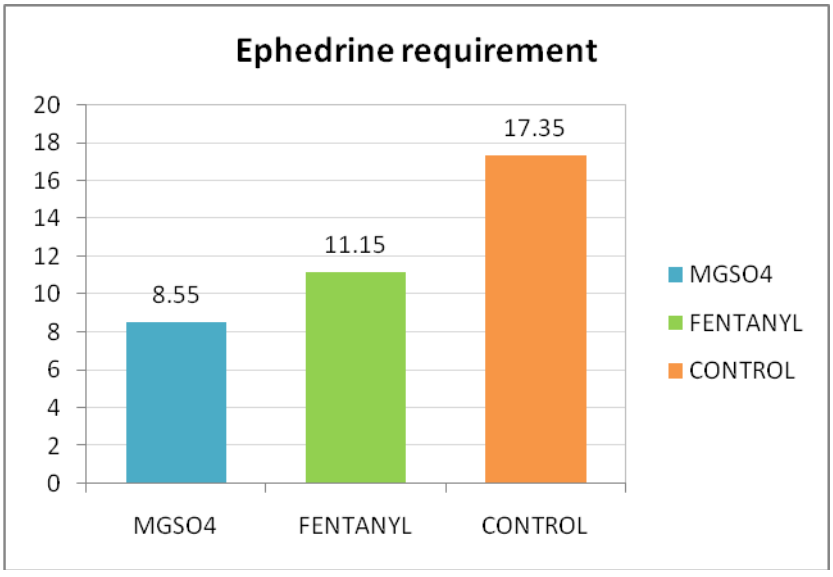
Ephedrine requirement in mg

	N	Mean	Std. Deviati	Std. Erro	95% Confidence Interv Mean		Minimun	Maximur
					Lower Bour	Upper Bour		
FENTANYL	20	11.15	3.297	.737	9.61	12.69	6	18
CONTROL	20	17.35	3.407	.762	15.76	18.94	12	24
$MgSO_4$	20	8.55	2.438	.545	7.41	9.69	6	12
Total	60	12.35	4.797	.619	11.11	13.59	6	24

ANOVA

Ephedrine requirement in mg

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	817.600	2	408.800	43.147	.000
Within Groups	540.050	57	9.475		
Total	1357.650	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

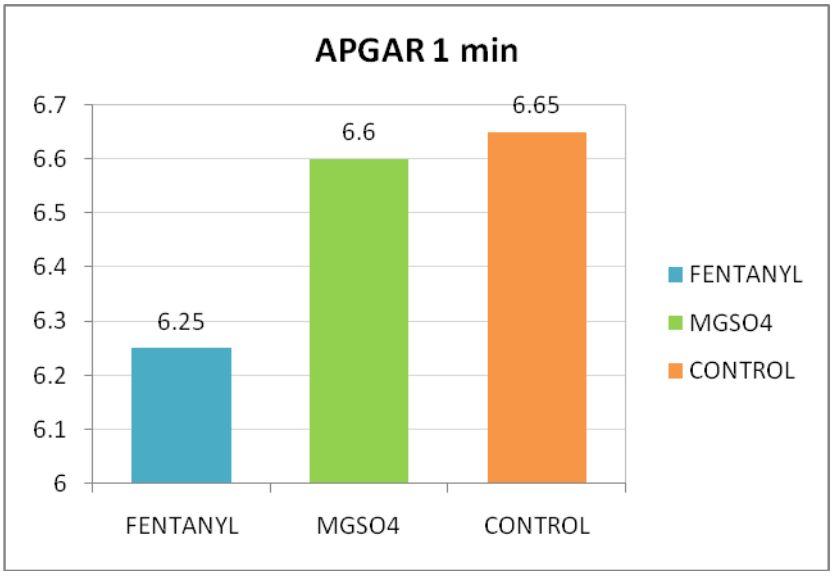
APGAR 1 min

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval f Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
FENTANYL	20	6.25	.716	.160	5.91	6.59	5	7
CONTROL	20	6.65	.933	.209	6.21	7.09	5	8
$MgSO_4$	20	6.60	.598	.134	6.32	6.88	6	8
Total	60	6.50	.770	.099	6.30	6.70	5	8

ANOVA

APGAR 1 min

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	1.900	2	.950	1.636	.204
Within Groups	33.100	57	.581		
Total	35.000	59			



Univariate Analysis, ANOVA & Two Group T-Test

Descriptives

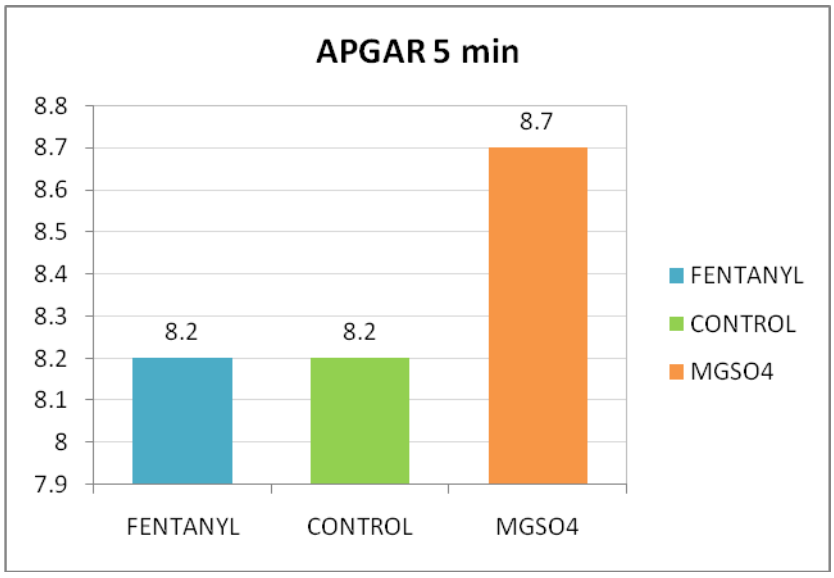
APGAR 5 min

	N	Mean	Std. Deviatic	Std. Erro	95% Confidence Interva Mean		Minimum	Maximun
					Lower Boun	Upper Boun		
FENTANYL	20	8.20	.951	.213	7.75	8.65	6	9
CONTROL	20	8.20	.768	.172	7.84	8.56	7	9
$MgSO_4$	20	8.70	.571	.128	8.43	8.97	7	9
Total	60	8.37	.802	.104	8.16	8.57	6	9

ANOVA

APGAR 5 min

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	3.333	2	1.667	2.746	.073
Within Groups	34.600	57	.607		
Total	37.933	59			



DISCUSSION

The study was conducted at Govt. Kilpauk Medical College on 60 pregnant patient with mild PIH ASA I and II undergoing elective Caesarean Section under spinal anesthesia after obtaining informed consent. Sensory and motor block onset time, upper level of analgesia, Duration of analgesia and motor blockade and APGAR score hemodynamics between the groups were evaluated.

The safety of intrathecal magnesium sulphate administration in humans and animals have been established. **SIMPSON et al** and **KROIN et al** demonstrated in animals by their study that intrathecal magnesium sulphate has a safety profile.

OZALEVLI et al and BUVENDRAN et al demonstrated no deleterious effects in humans on administration of intrathecal magnesium sulphate in their study they used 50mg of $MgSO_4$. The dose of magnesium sulphate was based on data from a rat model of postoperative pain in which188 micrograms of intrathecal magnesium sulphate potentiated morphine antinociception done by **KROIN et al.** Based on the relative differences between human and rat CSF volume and body weight, the 188 microgram dose was conservatively extrapolated to 50 mg for humans.

In my study sensory block onset time in magnesium sulphate group is (1.03 min) compared to control group (0.54 min) which is statistically significant (P value 0.000). Delayed onset of sensory block in magnesium sulphate group[12].

Onset of motor block in magnesium sulphate group is (9.15 min) when compared to control group (3.31 min) which is statistically significant (P value 0.000).[6] Hence there is a delay in the onset of motor block.

Intensity of motor block in Magnesium sulphate group is 2.25 and in control group 95 which is statistically less significant (P value0.291).[14]

Analgesic and motor block duration is prolonged in magnesium sulphate group (189.40 min) when compared to control group which is statistically highly significant (P Value 0.000)[6,25]

Fall in blood pressure and requirement of ephedrine is more in the control group (17.35 mg) compared to Magnesium sulphate groups (8.55 mg) (P value 0.000) highly significant due to high level of blockade[25]

Sedation score of Fentanyl group is 2.20 compared to control group which is statistically significant (P value 0.000).[25]

Duration of post operative analgesia- Duration is prolonged in Magnesium sulphate group 403.65 min when compared with control group 222.45, statistically highly significant(P value 0.000) [24,6]

There is no difference in APGAR score 1 min and 5 min between the groups (P value 0.204, 0.073) respectively statistically insignificant.

3 patients in fentanyl groups complaint of prurities, 2 patients due to inadequate blockade converted to GA were excluded from the study.

CONCLUSION

There is a delay in the onset of sensory and motor blockade with the use magnesium sulphate group .However there is prolonged motor blockade and duration of analgesia overlaps well into the postoperative period .This is beneficial for the patient for post operative analgesia, APGAR score not affected between the groups.

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ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK, CHENNAI- 10.

Venue: PANAGAL HALL, KMC
Dt: 01.06.2010

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10

Sub: Ethical Committee project work - approved – regarding.
Ref: Lr.No.975/Audit/08 Dt. 01.06.2010.

With above reference, the Institutional Ethical committee meeting for the following students was conducted at out Institution on 01.06.2010.

Name	Topic
Dr.D.Jayakumar, III year Mch. (Surgical Oncology) PG	Usefulness of Image guided Biopsy in cancer Patients
Dr.P.Saravanan, III year Mch (Surgical Oncology) PG	Study in Government Royapettah Hospital regarding the experience with aggressive Fibromatosis from 1996 to Till date
Dr.C.Amutha, I year MD Micro Biology	Bacterial Profile of Neonatal Sepsis
Dr.J.Mehrunnissa, I year MD Microbiology	Bacteriological profile in burns unit and their antibiotic susceptibility pattern at KMCH, Chennai
Dr.P.Venkateswaran, M.S.ENT	Anatomical Variations of the osteomeatal complex as a cause of chronic sinusitis and its correlation with the surgical result following functional endoscopic sinus surgery.
Dr.A.Arunagiri, MCh (Urology) PG	Comparative study of efficacy of localization and Fragmentation of renal stone by USG and Fluroscopy Guided ESWL.
Dr.J.Sivabalan, Mch Urology	Outcome of tension free trans obturator tape for female stress urinary incontinence.

Dr.N.Dinesh Kumar. PG Paediatrics	To asses the role of parentral steroids on the clinical course and outcome of Meconium Aspiration Syndrome in newborn.
Dr.A.Karthik. PG Anaesthesiology	Preinduction IV labetalol in attenuating intubation Response.
Dr.J.Rajaram. PG Anaesthesiology	Synergistic effect between dexmedetomidine and 0.75% Ropivacaine in Epidural.
Dr.K.Venkatesan. PG Anaesthesiology	Magnesium sulphate as an adjuvant to Intrathecal Bupivacaine in patients with mild preeclampsia undergoing caesarean section – Depth of Anaesthesia.

We are glad to inform you that at the Ethical Committee meeting the documents were discussed and the above short term projects are Ethically approved.

9/6/10

CHAIRPERSON

Prof. Dr.V.KANAGASABAI, MD.,

Dean

Govt. Kilpauk Medical College.

Chennai-10.

To

The Individuals

MASTER CHART

NAME: AGE/SEX: IP NO:

DATE: Ht.: GROUP:

DIAGNOSIS: Wt:

SURGERY:

COEXISTING ILLNESS:

EXAMINATION:

PR: CVS:

BP: RS:

RR: AIRWAY:

INVESTIGATIONS:

Hb: BLOOD UREA: HIV

URINE ALB: Sr. CREATININE: VDRL

SUGAR: BLOOD SUGAR: HBSAG

LFT

ECG:

BLOOD GROUP:

ANESTHESIA DETAILS:

PREMEDICATION:

LEVEL OF SAB

NO.OF ATTEMPTS:

DRUG DETAILS:

PATIENT DRUG HISTORY:

SAB GIVE AT:

PARAMETERS OBSERVED

SENSORY BLOCK ONSET TIME:

T12	T10	T8	T7	T6	T4	T3	T2

UPPER LEVEL OF ANALGESIA:

ONSET AND INTENSITY OF MOTOR BLOCK:

ANALGESIC & MOTOR BLOCK DURATION:

TWO SEGMENT REG TIME:

T2	T3	T4	T6	T7	T8	T10	T12	L1

SEDATION SCORE:

SENSORY SCORE:

HEMO DYNAMICS:

PARAMETERS	1 min	5 mins	10 mins	15 mins	20 mins	30 mins	45 mins	1 Hr	1 ½ Hr	2 Hrs	3 Hrs
PR											
BP											
SpO_2											
R.R											
UO											

DURATION OF POST OP ANALGESIA:

DABY DETAILS:

GROUP C- CONTROL

S. No.	IP NO	Name	Group	AGE	Height in cm	Weight in Kg	Hb %	Urine Alb	Urine sugar	Serum urea in mg%	Sr. creatinine in mg%	Level of SAB	No.of attempts	Sensory Block onset time T12	T 10	T8	T 6	T 4	T3	T 2	T1	UPPER LEVEL OF ANALGESIA	ONSET & INT OF MOTOR BLOCKADE	MOTOR BLOCK ONSET TIME in min	TWO SEG. REG TIME T2	TWO SEG. REG T4	TWO SEG. REG T6	TWO SEG. REG T8	TWO SEG REG T10	TWO SEG. REG L1	ANALGESIC & MOTOR B DURATION in min	SEDATION SCORE
21	6537	AARTHI	CONTROL	29	155	86	11	+	-	22	1	L3-L4	1	0.5	1	2	3	4		5		T2	2	5	72	87	100	112	132	140	140	1
22	5462	VANITHA	CONTROL	28	158	72	13	+	-	21	0.8	L2-L3	1	0.5	1	1.5	2	2.5		3		T2	2	3.5	60	75	88	103	120	130	145	1
23	6170	RUKMANI	CONTROL	23	158	66	12	-	-	25	0.7	L2-L3	1	1	1.2	2	3	3.5		4	4.5	T1	2	4	70	88	102	120	132	140	140	1
24	5501	PARVATHI	CONTROL	24	165	80	11	-	-	24	0.9	L4-L5	1	0.5	1	1.8	2	3		3.8		T2	2	3.8	67	85	100	110	122	135	142	1
25	6169	VEDHAVALLI	CONTROL	22	155	75	10	-	-	18	0.7	L3-L4	1	0.5	1	2	2.5	3		4	5	T1	2	5	66	80	92	108	120	134	134	1
26	6368	RESHMA	CONTROL	23	154	70	10	-	-	28	0.8	L3-L4	2	0.5	1	2	2.5	3		4		T2	2	4	78	90	102	118	130	140	138	1
27	6335	VIDHYA	CONTROL	23	154	74	10	+	-	22	0.8	L3-L4	1	0.5	1	2	3	4			4	T1	2	4.5	66	80	92	108	120	132	142	1
28	6151	VEENA	CONTROL	33	152	65	11	-	-	24	0.7	L3-L4	1	0.5	1	1.8	2.5				3	T1	2	2.5	77	85	97	105	120	130	135	1
29	5520	SURYA	CONTROL	25	156	64	11	+		26	1	L3-L4	1	0.5	0.7	1	1.5	2		2.5		T2	2	3	69	89	104	109	113	118	124	1
30	5631	ANANTHI	CONTROL	28	158	66	11	-		28	1.1	L3-L4	2	0.5	1	1.5	2	2.5		3		T2	3	2	66	80	90	96	101	110	120	1
31	6601	VIJI	CONTROL	24	160	76	11	-	-	26	1.1	L4-L5	2	0.5	1	2	2.5	3		3.5		T2	1	3	66	80	90	96	102	110	126	1
32	5247	mohini	CONTROL	28	148	52	9.8	-	-	24	0.8	L3-L4	2	0.7	1	2	3	3.5	4	5	5.5	T2	2	3.5	65	74	82	86	92	108	136	1
33	5238	RAGHINI	CONTROL	31	149	59	10	-	-	24	0.6	L3-L4	1	0.5	1	2	2.5	3	4	5	5.5	T2	2	2.5	70	76	82	90	98	104	134	1
34	6168	malathi	CONTROL	24	157	55	10	-	-	24	0.8	L4-L5	1	0.5	1	2	3	3.5	4	4.5	5	t2	2	3	72	84	92	100	109	112	130	1
35	6521	PARVATHY	CONTROL	32	162	58	11	+	-	18	0.6	L2-L3	1	0.5	1	2.5	3	4	4.5	5	5.5	T2	2	2.5	74	82	92	96	100	104	132	1
36	5542	GOMATHY	CONTROL	23	156	65	9.6	-	-	26	1	L2-L3	1	0.7	1.5	2	2.5	3	3.7	4.5	6.5	T1	2	3	68	80	88	95	100	105	108	1
37	6530	MALLIGA	CONTROL	28	150	68	11	+	-	28	1	L3L4	2	0.5	1	1.5	1.7	2	2.5	3	3.5	T1	2	3.5	86	92	97	100	106	112	116	1
38	6699	HEMALATHA	CONTROL	28	162	70	9.6	-	-	26	0.8	L4L5	1	0.5	0.7	1	1.5	2	2.5	2.7	3.5	T1	1	2	66	72	80	85	92	96	102	1
39	6741	REVATHY	CONTROL	21	154	66	8.6	-	-	22	1	L2L3	1	0.5	1.5	2	2.5	3.5	4.5	5	3	T3	2	3	65	70	76	82	89	95	100	1
40	6628	PREMA	CONTROL	28	148	72	11	-	-	26	1.1	L2L3	1	0.5	1	1.5	2	2.5				T2	2	3	72	78	82	88	92	102	108	1

SENSORY SCORE	PR 0 MIN	PR 1 MIN	PR 5 MIN	PR 10 MIN	PR 15 MIN	PR 20 MIN	PR 25 MIN	PR 30 MIN	PR 45 MIN	PR 1HR.	PR 2 HR	BP SYS 0 min	BP Dia 0 m	BP SYS 1	BP DIAS 1	BP SYS 5	BP DIA 5	BP SYS10	BP DIA10	BP SYS15	BP DIA15	BP SYS20	BP DIA20	BP SYS25	BP DIA25	BP SYS30	BP DIA30	BP SYS45	BP DIA45	BP SYS60	BP DIA60	BP SYS120	BP DIA120	SPO20	SPO21	SPO25	SPO210	SPO215	SPO220	SPO225	SPO230	SPO245	SPO260
2	86	86	90	106	108	100	92	86	82	80	80	156	92	148	86	142	78	116	70	140	76	132	80	138	76	116	70	128	72	132	86	130	77	99	99	99	98	97	100	100	100	99	98
1	86	92	78	82	84	90	100	90	92	86	80	146	110	152	108	140	90	146	100	114	63	130	88	136	92	142	90	146	92	144	90	130	99	99	99	99	98	100	100		99	98	99
2	82	88	92	96	94	98	92	90	92	86	89	146	96	122	82	112	76	126	68	106	56	106	64	100	56	104	62	110	68	126	76	132	82	99	99	99	98	100	100	99	100	100	100
2	90	120	110	106	104	100	106	104	90	82	86	146	90	90	60	80	60	90	60	104	50	92	50	106	50	112	60	122	72	132	86	136	72	99	99	99	98	100	100	100	99	100	100
1	86	104	94	100	104	106	90	86	88	89	78	142	94	102	64	126	58	130	70	116	60	104	50	118	60	122	64	128	72	132	72	130	76	99	98	99	99	99	97	100	100	99	99
2	86	85	104	114	96	84	80	78	82	86	88	146	92	114	74	90	70	126	76	114	80	114	89	126	82	132	84	136	82	140	80	139	89	99	99	98	99	100	100	98	99	100	99
2	89	90	88	92	98	82	86	90	82	78	75	144	92	132	86	128	72	108	66	118	74	128	82	132	84	136	82	132	80	136	86	135	89	99	99	98	99	98	99	99	100	99	99
2	88	86	88	96	94	100	94	100	96	100	104	142	96	142	92	113	84	106	82	100	52	106	62	100	60	98	53	110	70	122	76	130	84	99	99	98	100	99	99	100	99	99	100
2	88	90	110	106	102	88	86	82	89	90	90	168	92	150	100	110	60	100	70	110	60	124	74	130	82	134	86	138	82	130	80	136	90	99	100	98	98	99	100	100	99	98	100
1	82	92	106	108	100	88	86	78	84	78	86	150	94	146	94	122	76	100	72	112	70	126	76	128	78	134	82	136	84	140	80	142	86	99	100	100	100	98	98	98	98	99	98
2	82	82	76	76	88	82	80	78	84	86	82	144	96	118	96	124	84	124	68	117	58	123	69	130	72	134	78	128	76	132	76	134	86	99	99	98	100	100	99	98	100	100	100
2	88	88	92	106	108	96	84	82	86	84	82	152	96	134	82	124	72	116	76	122	86	128	86	134	86	132	84	136	80	132	82	130	80	99	99	99	98	97	100	99	99	100	99
2	86	84	96	104	108	110	94	88	90	84	88	148	100	148	100	128	84	110	80	122	60	124	84	134	82	136	82	136	82	130	82	130	78	99	99	99	98	100	100	99	99	100	99
2	84	86	90	104	108	102	88	82	84	82	84	142	96	132	86	120	76	104	86	112	60	106	62	110	86	124	86	130	82	120	86	130	81	99	99	98	100	99	99	100	99	99	100
2	86	84	90	104	106	100	86	72	86	82	86	154	94	134	94	120	84	106	74	112	72	118	82	124	84	130	86	130	84	130	86	130	86	99	99	99	98	100	99	99	100	99	100
2	78	84	86	92	100	102	94	88	86	76	80	144	98	138	86	114	74	104	60	108	66	112	72	122	76	126	80	132	76	128	78	136	82	99	98	99	100	100	99	99	99	100	98
1	80	82	90	96	106	102	92	88	82	78	82	140	92	132	86	128	78	118	80	102	62	100	72	114	80	122	88	130	86	132	70	138	80	99	99	99	99	98	97	100	100	99	99
1	86	100	106	105	106	102	98	104	112	102	94	144	92	100	60	118	76	140	84	130	78	134	88	128	84	126	84	112	80	122	84	132	89	99	99	98	99	100	100	99	100	99	98
1	94	142	132	130	120	126	118	106	94	90	88	146	94	140	90	126	64	112	66	134	74	124	68	118	78	126	76	128	80	130	82	132	84	99	99	99	99	98	100	100	99	99	99
1	84	90	92	96	100	104	88	82	100	98	99	130	82	122	76	118	82	107	76	118	72	124	86	120	82	132	96	124	99	114	78	132	96	99	99	99	99	98	99	100	97	99	99

SPO2 120	RR 0	RR 1	RR 5	RR 10	RR 15	RR 20	RR 25	RR 30	RR 45	RR 60	RR 120	URINE OUT PUT in ml	EPHEDRINE REQ in mg	DURATION OF POSTOP ANALGESIA in min	BABY WT in kg	APGAR 1	APGAR 5
99	16	20	18	18	16	18	16	14	18	18	16	50	18	240	2.75	6	7
99	14	18	20	22	24	22	18	16	14	16	18	70	12	235	2.2	6	8
100	18	20	18	22	24	20	18	22	26	18	16	60	18	206	2.5	7	8
100	20	22	18	22	24	28	32	30	26	22	18	50	12	210	2.75	6	8
98	24	22	18	16	18	22	18	16	18	20	22	75	18	226	3.25	6	8
99	20	16	18	22	16	20	16	22	20	18	22	60	18	236	2.8	5	7
99	20	18	16	18	14	18	20	18	16	18	18	60	18	232	2.8	6	8
100	22	20	18	22	20	20	18	22	20	18	20	60	18	212	2.5	6	7
99	20	22	24	18	20	20	20	18	26	18	16	50	18	200	2.75	6	8
99	18	20	22	20	18	14	16	20	22	22	23	30	12	208	2.8	6	8
99	16	18	20	22	24	26	28	24	21	23	14	25	24	232	1.7	6	7
99	16	18	14	18	16	14	18	16	20	22	18	60	18	232	2.9	8	9
99	18	14	16	18	20	14	22	14	16	22	24	50	18	238	2.8	8	9
100	22	20	18	16	18	14	16	14	18	16	22	50	18	214	2.5	8	9
99	21	18	16	14	16	15	18	16	14	18	18	45	18	232	2.7	8	9
97	21	16	18	22	17	23	14	18	20	20	22	40	20	214	2.65	8	9
99	23	16	17	20	22	21	24	26	22	22	21	30	18	226	2.8	7	9
98	24	16	17	18	24	20	18	21	18	20	22	60	15	216	2.5	7	9
99	16	22	24	24	26	23	18	20	18	19	22	25	12	198	2.75	6	8
99	16	16	181	20	22	24	28	26	22	18	18	30	24	242	2.8	7	9

GROUP F-FENTANYL

S. No.	IP NO	Name	Group	AGE	Height in cm	Weight in Kg	Hb %	Urine Alb	Urine sugar	Serum urea in mg%	Sr. creatinine in mg%	Level of SAB	No.of attempts	Sensory Block onset time T12	T 10	T8	T 6	T 4	T3	T 2	T1	UPPER LEVEL OF ANALGESIA	ONSET & INT OF MOTOR BLOCKADE	MOTOR BLOCK ONSET TIME in min	TWO SEG. REG TIME T2	TWO SEG. REG T4	TWO SEG. REG T6	TWO SEG. REG T8	TWO SEG. REG T10	TWO SEG. REG L1	ANALGESIC & MOTOR B DURATION in min	SEDATION SCORE
1	5649	revathy	FENTANYL	21	150	60	11	-	-	18	0.9	L4-L5	1	1	2	3	4	5		7		T2	2	4.5	122	138	150	165	182	186	142	3
2	5686	MOHANA	FENTANYL	23	160	86	11	-	-	17	0.7	L3-L4	1	0.5	1.5	2	2.5	3	7			T3	3	6	133	145	158	170	183	185	136	2
3	5676	sivagami	FENTANYL	26	148	64	11	+	-	20	0.8	L2-L3	1	0.5	1	2	3	4	5	6	8	T1	1	5	110	132	146	162	175	180	110	2
4	5486	UMA	FENTANYL	20	155	68	10	-	-	24	0.9	L4-L5	2	1	2	4	6	7	8	8.5		T2	2	6	95	110	122	135	148	150	132	2
5	5561	sumitha	FENTANYL	30	146	60	13	+	-	19	0.8	L2-L3	1	0.5	1	2	3	5	0			T4	2	3.5		120	134	148	160	165	140	2
6	6127	SUGUNA	FENTANYL	28	165	85	10	+	-	18	0.7	L4-L5	1	0.5	1	2	3	4,5	6	7	8	T1	3	5.5	108	120	132	146	161	163	134	2
7	6413	SIVASANKARI	FENTANYL	26	157	60	10	+	-	18	0.7	L3-L4	1	0.5	2	3	5	7	-	-		T4	2	4.5	128	140	153	170	180	180	112	2
8	6670	SUNDARI	FENTANYL	33	147	58	11	-	-	24	1.1	L2-L3	1	0.5	2	3.5	5.5	6.5	7	-		T3	2	5.5	-	132	145	168	170	183	136	3
9	6450	ANITHA	FENTANYL	26	150	66	11	+	-	28	0.8	L3-L4	1	0.5	1	2	3	4	5	6,5		T2	2	6.5	125	140	152	165	180	182	140	2
10	6642	SUGUNA	FENTANYL	28	153	68	12	+	-	20	0.9	L3-L4	1	0.5	1	4	6	8		6		T4	2	7		123	138	150	165	180	132	2
11	5750	DEVI	FENTANYL	28	148	64	9.4	+	-	18	0.9	L4-L5	1	0.7	1	2	3	4	5	7		T2	2	8	72	82	92	100	106	110	134	2
12	5758	JOTHI	FENTANYL	22	146	64	11	_	_	28	0.9	L3-L4	2	0.7	1	2	3	5	6			T3	3	6.5		72	82	90	96	101	138	2
13	6010	SUMATHY	FENTANYL	28	144	60	11	_	_	20	1	L2-L3	1	0.5	1	2	2.7	3	5	6		T2	2	4	74	86	95	102	107	112	114	2
14	5945	SUGANYA	FENTANYL	26	156	68	9.2	+	-	26	1.2	L4-L5	1	0.5	1	2	4.5	5	5.5	5.7		T2	3	5	66	74	82	87	92	97	124	2
15	6352	SHANTHI	FENTANYL	22	154	64	11	_	_	28	0.9	L3-L4	2	0.5	1	2	2.7	4.5	5.3	6		T2	2	4.5	70	79	88	94	100	118	136	2
16	5752	REKHA	FENTANYL	25	150	59	9.4	+		22	1.1	L2-L3	1	0.5	1	1.5	2	2.5	3.5	4.5	5	T1	2	6.5	92	100	106	110	116	122	136	3
17	7771	JEYAABBAS	FENTANYL	34	158	70	12	+	_	28	0.9	L4-L5	1	0.5	1	2	3	5	7			T3	2	5		130	142	156	162	168	140	2
18	6740	RADHA	FENTANYL	25	162	72	11	+	_	26	1.1	L4-L5	1	1	2	3	3.5	4	4.5		5	T1	2	3.5	74	86	95	102	107	112	114	2
19	6752	AMARAVATHI	FENTANYL	26	146	62	10			18	1.1	L2-L3	1	0.5	1	2	3	5				T4	2	3	110	122	130	135	140	146	156	2
20	6612	MAHESHWARI	FENTANYL	25	160	58	10			20	1	L3-L4	1	1	2	3	3.5	4	7			T3	2	7	92	100	106	110	116	122	132	3

SENSORY SCORE	PR 0 MIN	PR 1 MIN	PR 5 MIN	PR 10 MIN	PR 15 MIN	PR 20 MIN	PR 25 MIN	PR 30 MIN	PR 45 MIN	PR 1HR.	PR 2 HR	BP SYS 0 min	BP Dia 0 m	BP SYS1	BP DIAS1	BP SY S5	BP DIA5	BP SYS 10	BP DIA 10	BP SYS 15	BP DIA 15	BP SYS 20	BP DIA 20	BP SYS 25	BP DIA 25	BP SYS 30	BP DIA 30	BP SYS 45	BP DIA 45	BP SYS 60	BP DIA 60	BP SYS 120	BP DIA 120	SPO 20	SPO 21	SPO 25	SPO2 10	SPO2 15	SPO2 20	SPO2 25	SPO2 30	SPO2 45	SPO2 60
1	86	82	56	101	94	92	95	72	70	78	78	150	90	132	78	126	76	144	94	140	90	132	84	106	56	120	80	140	100	136	96	130	92	99	99	99	98	100	99	98	99	100	99
1	84	86	90	82	80	86	78	70	80	84	80	146	92	130	84	90	52	100	62	104	60	110	68	120	74	128	82	132	84	136	88	136	86	99	98	99	99	99	100	98	99	100	99
2	88	90	86	94	90	86	88	80	76	72	80	144	96	126	86	98	60	124	86	98	50	98	62	106	66	112	80	120	72	128	76	132	84	99	98	99	99	97	98	100	99	99	100
1	98	90	92	96	110	106	114	96	82	78	82	140	90	123	66	107	58	102	60	100	50	98	54	96	50	110	58	106	76	118	76	128	82	99	99	98	100	98	99	99	99	100	98
1	86	80	84	98	84	86	78	84	88	84	78	142	92	140	82	146	92	110	88	100	53	98	52	100	52	100	56	100	62	100	76	114	80	99	98	99	99	99	100	100	99	97	98
2	84	88	96	80	84	70	76	70	78	82	85	144	94	136	86	120	78	112	70	114	70	130	70	138	74	136	86	132	82	128	79	130	80	99	98	99	99	99	100	98	99	99	99
1	92	96	106	120	112	102	96	84	88	92	89	140	104	142	92	114	76	118	58	128	78	132	82	138	88	142	90	136	86	142	94	140	89	99	99	100	98	97	100	100	99	99	99
1	88	86	104	110	118	102	94	86	88	82	87	146	96	130	84	114	78	122	80	132	84	136	80	138	82	144	92	140	90	136	86	134	88	99	99	99	98	100	99	99	99	100	98
1	86	86	90	102	94	86	82	84	78	86	88	146	94	136	84	122	72	104	66	114	72	126	80	130	82	138	90	136	88	138	94	134	89	99	99	99	98	100	99	99	98	99	99
1	80	82	86	94	102	92	86	82	88	82	88	148	98	132	82	122	76	102	64	114	72	126	84	130	82	138	90	136	86	142	92	140	91	99	98	99	99	98	100	99	99	99	100
2	82	82	90	94	100	92	88	86	84	82	80	148	90	136	86	128	76	118	72	104	72	110	76	114	82	120	78	126	80	132	84	132	90	99	99	100	100	99	100	100	98	99	100
1	84	82	88	92	88	100	92	86	84	82	78	148	96	132	78	106	74	128	80	132	82	134	80	128	82	132	84	130	86	134	86	132	90	99	100	99	99	99	100	98	100	99	100
1	80	86	96	94	92	96	98	90	84	86	82	140	94	132	86	108	82	114	86	104	82	114	78	126	82	132	86	130	82	134	84	136	78	99	100	100	99	98	99	100	98	100	99
1	82	84	92	94	88	100	92	88	82	78	82	138	92	124	78	104	78	114	76	128	80	106	82	118	82	126	76	130	82	132	84	136	86	99	100	99	100	98	99	99	98	100	99
1	82	86	90	94	106	96	88	84	82	80	84	144	94	136	92	106	60	116	72	102	76	122	76	130	82	132	78	136	82	132	82	128	82	99	100	100	99	100	99	98	100	99	98
1	82	80	88	90	100	94	90	84	84	80	88	136	90	128	68	132	74	126	80	104	64	108	70	116	76	124	80	130	84	134	82	136	80	99	100	98	100	99	100	98	100	99	99
1	80	84	98	88	88	84	92	82	80	78	82	158	92	142	86	106	60	140	58	122	64	126	68	138	64	132	72	130	80	134	82	132	86	99	100	99	100	99	100	99	100	98	99
1	80	88	86	82	90	94	86	82	88	82	83	150	94	124	86	106	62	116	72	120	76	128	82	132	80	134	84	134	86	136	88	136	78	99	100	100	99	100	98	100	100	100	99
1	82	82	56	101	96	94	95	72	71	80	82	152	92	122	88	126	76	132	82	136	84	106	56	102	62	120	82	130	86	132	84	134	86	99	100	99	98	100	100	100	99	98	99
1	82	78	86	92	96	100	92	86	82	84	82	136	90	128	68	132	74	126	80	104	64	108	70	116	76	124	80	130	84	134	82	136	80	99	100	98	100	99	100	98	100	99	99

SPO2 120	RR 0	RR 1	RR 5	RR 10	RR 15	RR 20	RR 25	RR 30	RR 45	RR 60	RR 120	URINE OUT PUT in ml	EPHEDRINE REQ in mg	DURATION OF POSTOP ANALGESIA in min	BABY WT in kg	APGAR 1	APGAR 5
99	16	18	20	24	22	18	16	20	17	18	16	80	12	334	2.75	7	9
99	18	16	20	22	24	20	22	16	18	14	16	70	12	322	2.7	6	8
99	16	16	20	24	28	26	20	18	18	16	20	50	8	262	2.4	7	9
99	16	18	22	20	22	22	20	18	20	16	18	60	12	273	2.3	5	6
99	16	18	20	18	20	20	22	24	18	16	20	80	6	296	2.6	6	8
99	16	16	18	24	16	20	18	16	22	20	19	75	12	336	2.5	6	7
99	16	16	18	20	18	16	20	22	18	16	18	60	12	296	2.75	6	7
99	16	16	18	20	18	16	20	22	18	16	18	60	9	310	2.4	5	7
99	16	20	18	22	24	20	18	16	20	22	20	50	12	276	2.4	6	8
100	18	14	16	20	18	20	24	18	20	22	21	50	18	286	3.4	6	8
100	16	18	17	20	22	21	19	17	18	17	16	50	12	318	2.8	7	9
99	16	16	18	19	20	22	20	24	18	18	16	60	12	334	2.8	7	9
100	16	16	18	20	21	20	22	24	18	19	20	30	6	296	2.3	7	9
98	16	16	18	20	21	22	18	19	20	22	20	50	12	338	3	7	9
99	16	18	20	19	22	19	20	21	22	24	20	40	8	280	3	7	9
99	16	18	14	18	17	20	22	24	26	22	19	50	12	336	2.7	6	9
99	19	16	18	24	28	22	20	18	22	24	21	30	6	338	2.3	6	9
99	18	16	18	20	22	24	26	28	24	22	20	50	12	336	3	5	7
100	18	16	18	20	24	28	22	24	20	18	16	30	12	316	3	7	9
99	18	14	16	17	20	22	24	26	22	29	20	40	18	333	2.7	6	8

GROUP M- $MgSO_4$

S. No.	IP NO	Name	Group	AGE	Height in cm	Weight in Kg	Hb %	Urine Alb	Urine sugar	Serum urea in mg%	Sr. creatinine in mg%	Level of SAB	No.of attempts	Sensory Block onset time T12	T 10	T8	T 6	T 4	T3	T 2	T1	UPPER LEVEL OF ANALGESIA	ONSET & INT OF MOTOR BLOCKADE	MOTOR BLOCK ONSET TIME in min	TWO SEG. REG TIME T2	TWO SEG. REG T4	TWO SEG. REG T6	TWO SEG. REG T8	TWO SEG REG T10	TWO SEG. REG L1	ANALGESIC & MOTOR B DURATION in min	SEDATION SCORE
41	6155	REKHA	MGSO4	22	155	68	98	+	-	18	0.8	L3L4	1	1	2.5	3.5	5	7.5				T4	2	7	110	134	142	150	160	165	190	2
42	5483	ARULMANI	MGSO4	25	152	70	9.8	-	-	26	0.8	l3l4	1	1	2	3	5	8				t4	2	8	122	136	142	152	160	171	206	2
43	5430	laisa	MGSO4	25	150	70	12	+	-	22	0.9	l3l4	1	0.5	1.5	2.5	4	6	8	10		t2	2	11	120	136	142	150	162	166	206	2
44	5990	janaki	MGSO4	21	148	65	11	-	-	22	0.9	L2L3	1	1	3	5	7	9.5				T4	2	6.5	120	134	140	156	166	170	162	2
45	6076	SARANYA	MGSO4	22	146	66	9.8	-	-	15	0.6	l2l3	2	1	2	3	5	10				t4	2	10	116	130	140	145	152	162	156	1
46	4799	MARY	MGSO4	24	156	54	10	+		24	0.9	L3L4	1	1	2	4	5	7	9			T2	2	9	106	120	130	142	147	156	166	2
47	5619	SANGEETHA	MGSO4	18	160	62	8.2	+		20	0.9	L4-L5	1	2	3	4	5	6	9	12		T2	3	12	120	132	140	155	160	172	210	2
48	5417	KAVITHA	MGSO4	27	148	78	10	+	_	50	0.8	L2L3	1	1	2	3	4	5		8		T2	3	7	106	120	132	140	152	160	204	3
49	6013	UDAYALAKSHMI	MGSO4	24	152	56	11	+	_	29	0.9	L3L4	1	0.5	1	2	5					T6	2	10	106	120	126	135	142	150	186	3
50	5721	BHARATHI	MGSO4	24	160	60	10			29	0.8	L3L4	1	1	2	3	4	6				T2	3	9	102	122	128	132	142	148	170	2
51	6652	SHANTHI	MGSO4	24	150	70	11	+	_	42	0.7	L3L4	1	0.5	1	1.7	3	3.7				T4	2	9	94	122	140	150	156	158	202	2
52	6651	VAANI	MGSO4	28	162	72	9	+	_	26	1	L4-L5	1	2	3.5	4.5	5.5	6.5	8			T3	3	8	92	106	112	120	126	132	210	2
53	6282	TAMILARASI	MGSO4	20	160	72	11	+	_	22	1	L4-L5	1	1	2	3.5	4	5	8			T4	2	8	90	102	110	118	126	130	210	2
54	6632	MALA	MGSO4	28	156	66	11	_	_	24	0.9	L3L4	1	1	2	2.5	3.5	4.5	8.5			T2	2	8.5	94	108	116	128	130	136	174	1
55	6542	MANONMANI	MGSO4	32	152	57	11	_	_	28	0.8	L3L4	2	1	2	3	4	6				T2	2	9	90	105	114	120	125	130	160	1
56	6756	SAROJA	MGSO4	23	164	74	12	+	_	22	1	L4-L5	1	1.5	2.5	4	6	8				T6	2	8				98	110	122	218	2
57	6759	SARANYA	MGSO4	22	156	65	8.8			24	0.9	L3L4	1				3	4	5	6		T3	2	9	76	89	99	111	121	134	192	2
58	6261	PRABHA	MGSO4	27	145	58	11			18	0.9	L2L3	1	1	2	3.5	7	9	11	12		T3	1	11	88	90	98	100	102	112	186	2
59	6226	KASTHURI	MGSO4	32	158	72	12			22	0.9	L2L3	1	1	2	4	6	8	9	10		T4	2	11	90	99	105	109	124	129	199	2
60	6458	AMUDHA	MGSO4	28	160	60	9.8			18	0.8	L4-L5	1	0.5	2	3	5	7	9	12		T3	2	12	93	107	114	119	129	132	181	2

SENSORY SCORE	PR 0 MIN	PR 1 MIN	PR 5 MIN	PR 10 MIN	PR 15 MIN	PR 20 MIN	PR 25 MIN	PR 30 MIN	PR 45 MIN	PR 1HR.	PR 2 HR	BP SYS 0 min	BP Dia 0 m	BP SYS 1	BP DIAS 1	BP SYS 5	BP DIA 5	BP SYS10	BP DIA10	BP SYS15	BP DIA15	BP SYS20	BP DIA20	BP SYS25	BP DIA25	BP SYS30	BP DIA30	BP SYS45	BP DIA45	BP SYS60	BP DIA60	BP SYS120	BP DIA120	SPO20	SPO 21	SP O25	SPO210	SPO215	SPO220	SPO225	SPO230	SPO245	SPO260
1	92	94	96	67	72	78	72	78	82	92	94	146	92	124	96	130	95	127	82	132	72	136	80	140	82	146	90	132	82	134	90	136	86	99	99	99	98	97	100	99	99	99	99
2	78	112	112	86	110	106	94	92	88	82	80	148	86	140	100	118	76	110	70	106	61	112	70	110	62	110	70	118	70	122	74	136	76	99	99	99	99	99	99	99	100	98	97
1	86	80	80	100	100	90	96	92	88	84	86	140	100	110	80	120	80	110	80	110	70	110	80	1112	80	114	76	122	80	132	86	132	82	99	99	99	98	100	100	99	99	99	99
2	80	92	98	103	96	92	98	100	96	92	84	160	100	154	92	126	80	133	79	122	84	112	60	110	64	116	80	126	82	132	86	134	89	99	99	99	98	99	100	99	98	97	99
1	98	88	95	98	89	91	89	88	86	84	88	144	96	132	96	115	78	131	80	128	84	118	72	108	68	112	72	118	76	126	88	134	80	99	98	99	99	100	99	98	99	99	99
3	88	88	90	68	74	80	78	84	82	76	87	144	92	140	92	100	60	80	56	100	60	106	72	104	68	110	70	122	76	126	82	132	82	99	98	99	99	99	99	99	99	99	99
1	82	78	76	100	92	88	80	88	76	72	86	142	94	148	94	146	96	140	86	130	76	122	62	122	62	140	80	136	80	132	86	132	86	99	98	100	99	99	99	99	99	100	98
1	82	86	82	98	100	86	84	84	76	84	102	142	96	130	80	125	72	111	70	130	80	132	84	128	84	138	86	144	90	132	88	134	86	99	98	98	98	99	100	99	99	100	99
1	86	82	76	88	92	88	82	84	86	88	78	139	90	140	90	132	72	124	78	109	56	109	72	114	82	120	82	124	86	136	88	134	88	99	99	99	98	100	100	99	99	99	99
1	92	112	88	94	98	88	82	86	86	82	82	144	92	118	90	128	84	108	80	120	70	118	68	114	74	116	78	114	66	107	74	132	86	99	98	100	99	98	100	100	100	100	100
1	84	78	84	82	88	90	86	78	82	82	80	136	96	98	60	124	84	114	64	100	58	102	62	115	70	116	80	116	82	122	82	134	81	99	99	99	98	99	99	98	99	98	99
2	88	86	92	96	92	98	100	86	82	80	78	144	96	128	82	132	82	130	84	132	82	122	76	130	80	132	76	134	80	136	86	132	86	99	98	100	99	99	99	99	99	99	98
2	92	86	82	78	82	86	78	80	82	78	84	136	96	124	76	114	82	118	76	1126	78	128	80	122	72	132	84	132	78	136	86	138	80	99	99	99	100	98	98	98	99	99	99
2	86	92	104	108	94	96	88	87	84	86	88	148	96	144	96	130	84	116	86	118	74	120	84	116	84	118	84	120	80	120	84	116	84	99	99	98	99	99	98	99	98	98	99
2	90	96	104	100	96	84	86	88	90	94	96	140	100	136	94	120	84	124	69	114	79	124	78	118	84	120	86	118	85	134	87	132	84	99	99	98	98	97	100	99	99	99	99
2	96	102	98	92	88	84	86	82	78	80	82	138	90	132	86	126	80	122	80	124	80	126	80	128	82	130	82	138	78	130	84	136	86	99	98	99	99	99	98	99	99	99	99
1	116	108	88	88	96	84	80	82	78	80	74	138	92	128	86	100	60	122	60	120	70	120	82	130	70	116	76	108	82	110	70	112	66	99	99	98	100	99	99	99	98	99	99
1	80	86	98	95	94	92	99	79	89	78	83	139	90	112	68	122	70	126	87	128	68	132	80	132	87	122	78	143	90	132	68	124	87	99	99	99	99	99	98	100	99	100	98
2	78	76	82	99	97	92	94	78	83	82	88	143	94	112	80	124	80	124	76	132	80	128	90	122	90	134	87	132	81	130	80	138	93	98	100	100	100	99	98	99	99	99	99
1	92	91	92	86	88	82	93	81	82	84	86	136	92	113	70	124	78	134	87	129	86	132	80	132	80	128	78	124	78	137	92	136	80	99	98	99	98	99	99	99	99	99	99

SPO2 120	RR 0	RR 1	RR 5	RR 10	RR 15	RR 20	RR 25	RR 30	RR 45	RR 60	RR 120	URINE OUT PUT in ml	EPHEDRINE REQ in mg	DURATION OF POSTOP ANALGESIA in min	BABY WT in kg	APGAR 1	APGAR 5
99	14	16	24	28	28	22	20	18	16	20	22	60	6	394	2.5	7	9
100	15	18	22	18	20	22	20	24	22	18	20	50	9	382	3.5	6	8
98	16	16	18	20	16	22	20	18	20	22	22	50	12	376	2.7	6	8
99	18	20	18	18	22	20	20	22	16	18	24	80	6	420	3.75	6	8
99	19	22	18	18	20	15	20	22	24	22	20	40	9	386	2.8	7	9
99	18	14	16	18	18	14	20	22	22	24	24	40	9	456	4.6	7	9
99	14	16	18	19	18	19	20	20	22	22	24	50	6	380	2.8	6	8
99	14	16	18	22	18	18	20	22	20	22	22	40	12	426	2.75	6	9
99	16	18	18	19	15	16	17	22	22	22	22	60	9	396	2.5	6	9
99	18	18	19	17	19	20	20	20	20	20	24	50	9	400	2.8	7	9
99	18	20	19	17	16	14	20	23	24	22	22	40	6	388	3.25	6	7
99	18	17	19	16	18	19	18	18	18	18	17	30	12	392	2.9	7	9
99	20	16	19	19	18	17	16	15	14	16	18	40	6	446	2.95	7	9
99	16	1	18	19	18	18	18	16	17	18	18	50	9	408	2.8	8	9
99	18	18	16	15	14	20	20	25	25	25	26	60	12	428	3.1	7	9
99	18	17	17	16	15	14	17	18	18	24	22	40	6	460	2.76	7	9
99	15	16	18	17	13	15	17	17	18	24	22	50	12	406	3.25	6	9
100	23	24	26	28	23	24	25	26	22	24	25	30	6	382	2.6	7	9
99	22	16	18	19	20	22	23	24	25	26	27	70	6	376	2.9	6	9
99	21	22	23	25	26	28	22	21	23	22	21	80	9	371	2.7	7	9